THE VENOUS AND LIVER PULSES, AND THE ARHYTHMIC CONTRACTION OF THE CARDIAC CAVITIES.

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So far as our clinical methods of examination are applicable to the study of the heart they nearly all have reference to the condition of the left side of the heart. And yet, if carefully sought, symptoms connected with the right side are of no infrequent occurrence, did we but possess the means for observing them. Of these symptoms, the most striking and the most instructive is the pulsation in the veins, which, in a sense, bears a relation to the right heart comparable to that which the arterial pulse bears to the left. The venous pulse, however, offers to us a different kind of knowledge, inasmuch as it is itself a pathological manifestation which gives information regarding the condition of two cavities of the heart (the right auricle and ventricle), the arterial pulse affording direct knowledge of the left ventricle only. The venous pulse, too, presents a greater variety of features, and is subject to influences so subtle that it may manifest variations with the changing condition of the patient, during which the arterial pulse reveals no appreciable alteration. Did we but fully comprehend the import of these variations, then would our knowledge enable us to understand and combat certain diseased conditions of the heart as yet very obscure and ill defined. To assist in clearing up this subject I have contributed a preliminary paper;[1] again approaching the subject purely from the clinical aspect as in the first instance, this investigation has been undertaken merely to understand certain obscure phenomena connected with sundry changes observed taking place in the circulation under a variety of conditions.

My attention was first directed to this subject as a result of a study of the changes that occur in the heart during pregnancy. It had been my misfortune to have a series of pregnancies, complicated by heart disease, ending disastrously, and I was naturally led to inquire into the state of the maternal circulation during that period. The usual descriptions given of these changes did not appear to me to be adequate, either as to

[1] *Journal of Pathology and Bacteriology*, 1892, vol. i. p. 53.

the strength of the arguments advanced or as to the interpretation of the so-called facts on which these arguments were based. I was so circumstanced that I had plenty of material for observation, and could follow individual cases for a long period through successive pregnancies and incidental illnesses, until their restoration to health. Making careful notes of every little circumstance, it soon became apparent that there were numerous symptoms of whose presence scant record had been made. Of these the venous pulse was the most striking. After noticing its forms I consulted such authorities as my limited opportunities at the time permitted me to have access to, and I found that the conclusions I had arrived at differed materially from those arrived at by the authors whose works I was able to consult. It seemed to me that if graphic records of the venous pulse, with a standard time, could be obtained, much light might be thrown upon the subject. Being myself devoid of all mechanical skill, my attempts were crude and tediously prolonged; and when at last I did succeed, it was found, as was to be expected, that both the method and the results had been already described. But it was fortunate that I was ignorant of the work that had been done, for had I been aware of what so many skilled observers, with the advantage of experimental research, had accomplished, I should have been deterred from attempting, at such great disadvantage, to follow them, and should have accepted their interpretations without question. As it was, in my numerous attempts to get a graphic record I acquired an intimate knowledge of the strength, the variations, and the situation of the pulsations in the veins. When, at last, I did get sufficient results, unfettered by any prejudiced notion of what should happen, or what others had explained, I evolved the interpretation given in the following pages. Here likewise, to a great extent, I had been anticipated, but my opinion still is that the most salient features of the more commonly occurring venous pulse had been entirely misunderstood by both clinical observers and experimental investigators. The subject, so far, has been involved in the greatest confusion, and has given rise to endless contradictory assertions and observations.

The question has really been, In what manner is moderate tricuspid incompetence manifested in the veins? To this no satisfactory answer has been returned, simply from the fact that *the influence of the dilating auricle between the right ventricle and the veins has been overlooked.* When considering my results, and carefully studying the factors at work, I reckoned on this, and found in it a fitting explanation for the results I had obtained. In the course of my inquiry into this subject I have read all the literature available to me, and in only one instance has this factor been hinted at. I am indebted to Dr. Wilks for calling my attention to the work of his old master, Wilkinson King, whose careful investigations, experimental and morphological, into the question of tricuspid incompetence are a model of patient research and logical deduction. King states that the reason the venous pulse is not

observed as a common result of tricuspid incompetence is that the overplus is taken up by the capacious auricle and vena cava.[1] The means at his command did not permit the demonstration of the manner in which this modifying influence takes place.

By the method I have now devised—the use of the clinical polygraph—the movements of the veins and the time of occurrence of their movements can be readily demonstrated, and the causes of each individual movement inferred. Hitherto great varieties of movements have been described, and a great variety of causes as producing them. It will be found that all these varieties are simplified, and that even mere vibratory tremors can be registered, that each vibration has a distinct and characteristic significance, and that its cause can readily be recognised (Figs. 76 and 108). Recognising, then, more accurately, each individual feature, the variations with the changing conditions of the heart form an interesting and somewhat complex study, more particularly when there is distinct evidence of the lack of rhythm between the actions of the various chambers of the heart. Clear and distinct as the movements constituting the venous pulse are, there are still many features left unexplained. The subject is one of greater proportions, and affords opportunities for more careful observation than one would at first imagine. The venous pulse being so susceptible to the most subtle changes in the circulatory and respiratory systems affords evidence of the most delicate type of any influence affecting those systems. A few of these changes only can be indicated in this paper, but they are such as to lead one to hope that, with a more accurate knowledge of the subject, a comparatively wide field for clinical observation will be opened up.

In drawing up this paper I have been careful to avoid merely the registration of exceptional cases, but have extended my observations over a large number of patients (about 500), and under a great variety of conditions. The explanation given of the features of that form of pulse called the auricular form, which is by far the most common, has been as applicable to every case as it is to those recorded in this paper. Usually, however, the features in cases presenting the form of pulse called the ventricular, have individual peculiarities of which those here recorded may be taken as examples. If care be taken to have the venous pulse registered at the same time as the arterial pulse (carotid or radial), little difficulty is experienced in determining the nature of each feature.

Whilst I shall frequently have occasion to refer to the erroneous views of others, I am conscious that where so many more competent observers have erred, it is but likely that I should go astray. The necessity, therefore, has been kept distinctly in view in composing this paper, of giving a clear conception of the facts, as distinct from the

[1] King, T. W., "The Safety-Valve Function in the Right Ventricle of the Human Heart," *Guy's Hospital Reports*, 1837, vol. ii. p. 132.

theories; on that account numerous illustrations have been used, and the manner of taking the tracings and of timing the events has been fully explained, so that should the interpretations be valueless, the recorded movements of the veins may still be reliable, and may serve for other and more fitting explanations. In like manner, the cases incorporated in the text are given in order to indicate the conditions under which the various forms of the venous pulse occur, and the symptoms noted are mainly those that serve to indicate the nature of the patient's condition with reference to the circulatory system.

I have not attempted to draw any conclusions from the result of the inquiry into the action of the heart chambers during periods of irregular rhythm. It would seem that a great variety of forms may arise, but the cases here given are typical of those that have come under my observation. I have been content, therefore, to place on record a few of those that I have observed, and to give a classification of them in the summary. Hitherto the causation of the various events in the venous pulse being obscure, it has not been possible to draw reliable deductions from the cases recorded.

It has been deemed advisable to preface the subject proper with a brief historical introduction, more for the purpose of indicating how the knowledge of this subject has been gradually acquired, than of attempting a complete record of what has been done on this subject in the past. Additional historical references will be incidentally alluded to.

The subject will be dealt with in the following order:—

Section I.—Historical Introduction.

1. *The Venous Pulse.*—From incidental notices various of the older writers seem to have been conversant with the pulsations in the veins, particularly of those of the neck. The first authentically reported case appears to have been made by Homberg in 1704.[1] So far as one can understand the description given by him, the pulsations occurred in a patient suffering from great cardiac dilatation, in whom violent attacks of asthma (dyspnœa ?) occurred, during which the pulsations in the veins were developed. At the post-mortem examination the cavities of the heart and a portion of the adjacent arteries contained "polypi," and the author attributed the venous pulse to the backward discharge of blood, in consequence of the obstruction offered by the polypi. Homberg also made a very interesting observation, namely, that the pulsations in the veins corresponded to the beats of the heart, while the pulse did not—a fact with which this paper will be considerably concerned. Lancisi[2] observed the exaggerated venous pulse in chlorotic females after exertion, but distinguished it from the more serious form by the fact that it disappeared when the patient was quiet. Did it persist even then he assumed it to be a symptom of dilatation of the right heart with incompetence of the tricuspid valves. Morgagni[3] not only recognised the venous pulse but distinguished, for the first time, the fact of a pulsation being due to the auricle—timing the venous pulse by the temporal artery pulse. If it were synchronous with the arterial pulse, and therefore due to the action of the ventricles, he considered it might be due to the vehement driving back of the valves (a view afterwards revived by Friedreich) or to incompetence of the valves. He was not aware, however, of the frequency of the auricular venous pulse, and assumed it to be due to very grave forms of heart disease. Senac[4] accepted Homberg's description, but disputed Morgagni's observation regarding the auricular pulsation, and asserted that there could only be a stasis of blood, holding that the jugular valves would prevent any regurgitation, while Corvisart[5] maintained, evidently, that there is no such thing as pulsation in the veins—the previous observers having been led astray by the carotid

[1] "Histoire de l'Academie Royale des Sciences," Année 1704, Paris, 2nd edition, 1722.

[2] Lancisi, M., "De Motu Cordis et Aneurysmatibus." Opus Posthumum. Lugd. Bat. 1740.

[3] Morgagni, J. B., "De Sedibus et Causis Morborum." Translated by G. B. Alexander. London, 1769. Book ii. Letter xviii. Article 9.

[4] Senac, J., "Traité de la structure du Cœur, de son action et de ses maladies," Paris, 1783, 2nd edition, tome ii. p. 476.

[5] Corvisart, J. M., "Essai sur les maladies et les lésions organiques du Cœur et des gros vaisseaux," Paris, 1818, 3rd edition, pp. 137 and 149.

pulse. Burns,[1] however, agrees with Morgagni as to the occurrence of an auricular pulse. Although it was asserted by the older physiologists, such as Haller[2] and Bertin,[3] that the auricle did discharge the blood backward, under certain circumstances, little credence was given to the power of the auricle in this respect by most clinical observers. Bremmer,[4] however, insisted on the fact that the auricle produced this pulsation, and Skoda related a case where it occurred. Bamberger,[5] commenting upon Skoda's case, states that he had never observed a venous pulse under the conditions described, while Hamernjk[6] is at great pains to show that the observation must have been erroneous, as such a thing as a pulsation in the veins during the auricular systole could not possibly happen. In the writings of authors during the earlier half of this century there is usually only one form of venous pulse recognised—the time of its occurrence is not generally explained, but is evidently assumed to be synchronous with, and caused by, the ventricular systole. Discrimination was made between pulsating swelling, undulations, oscillations, vibrating tremors, regurgitant waves, and turgescence without movement (Kreysig,[7] Adams,[8] Williams,[9] Hope,[10] Bouillaud,[11] Laennec,[12] Beau,[13] etc.), while in rare instances a difference was observed between the rate of the venous pulse and that of the radial artery.[14]

During the earlier decades of this century a number of cases of pulsation in the more peripheral veins were recorded, and much

[1] Burns, Allan, "Observations on some of the most frequent Diseases of the Heart," etc., Edinburgh, 1809, p. 129.

[2] Haller, A., "Lectures on Physiology." Translated by S. Mihles. London, 1754.

[3] Bertin, M., "Mémoire sur la principale cause du gonflement et du dégonflement-alternative des veines jugulaires, etc.," "Histoire de l'Academie Royale des Sciences," Année 1763, p. 260.

[4] Bremmer, J. A., "De Causis Pulsum Arteriarum Venarumque inducentibus," Inaug. Diss. Dorpat, 1831.

[5] Bamberger, H., "Lehrbuch des Herzens," Wien, 1857, p. 100.

[6] Hamernjk, J., "Untersuchungen ueber die Erscheinungen an den Arterien und Venen," etc., Prag. 1847, p. 244.

[7] Kreysig, F. L., "Die Krankheiten des Herzens." Opus posthumum. Edited by O. Hohlschnitter. Berlin, 1845, p. 146.

[8] Adams, R., "Cases of Diseases of the Heart," etc., *Dublin Hospital Reports*, 1827, vol. iv. p. 353.

[9] Williams, C. J. B., "The Pathology and Diagnosis of Diseases of the Chest," London, 1835, 3rd edition, p. 182.

[10] Hope, J., "A Treatise on the Diseases of the Heart and Great Vessels," 3rd edition, London, 1839, pp. 309, 543, 545, etc.

[11] Bouillaud, J., "Traité clinique des maladies du Cœur," Paris, 1835, tome ii. obs. 124, p. 395, etc.

[12] Laennec, R. T. H., "A Treatise on Mediate Auscultation and the Diseases of the Lung and Heart," edited by T. Herbert, London, 1846, pp. 578 and 581, etc.

[13] Beau, J. H. S., "Traité expérimental et clinique d'auscultation appliquée à l'étude des maladies du poumon et du cœur," Paris, 1856, p. 320.

[14] Adams, *loc. cit.* p. 426. Also Pearson, G., "The Substance of a Clinical Lecture on the Diseased Valves of the Heart, producing Pulsation in the Jugular Veins," *Edin. Med. and Surg. Journ.* 1816, p. 193.

discussion ensued as to the cause of this phenomenon. Besides the backward propulsion of blood from the right side of the heart there were described, as causes, the pulsatile movement of the arteries communicated through the capillaries to the veins, the independent contractility of the veins themselves (Graves)[1] and—from a reference in Anke's[2] dissertation—even a magnetic force. A number of these cases, such as those of Steinbuch,[3] Sundelin,[4] Davis,[5] and Benson,[6] appear to have been regurgitant from the right heart, while other cases reported by Elliotson,[7] Beyer,[8] Ward,[9] and Graves[10] appear to have been caused otherwise. The possibility of a propagation of the arterial intermittency through the capillaries to the veins was demonstrated by Palmer[11] in the veins of a frog's legs by obstructing the flow—the pulsation of the arteries being transmitted through the capillaries to that portion of the distended veins distal to the point of compression. Later Bernard's well-known experiment showed that on stimulation of the chorda tympani the arterioles of the salivary gland became so dilated that the arterial pulse was transmitted to the veins.[12] Gradually this feature in the venous pulse became recognised, and Quincke[13] and Broadbent[14] have shown that the conditions necessary for its production are great arterial relaxation. It occurs most frequently in cases of aortic incompetence, but other conditions that give rise to dilatation of the smaller arteries, such as chlorosis and neurasthenia,[15] may also produce it. It may occur associated with no disease, and its presence is even consistent with perfect

[1] Graves, R. J., "On Pulsation of the Jugular Veins," *Lond. Med. Record*, 1831, vol. vii. p. 550.

[2] Anke, N., "Dic vitiis nonnullis rarioribus cordis observationes quædam," Inang. Diss. Dorpat, 1832.

[3] Steinbuch, "Ein Beitrag. zur Gründung einer wissenschaftlichten Kenntniss des Pulsschlages," *Journal der praktischen Heilkunde*. Berlin, September 1815.

[4] Sundelin, "Ueber die Schwerigkeit der Erkenntniss innerlicher sogenannter organischer Fehler; nebst einiger wichtige Krankengeschichten," *Archiv f. med. Erfahrung*, Berlin, July 1822, p. i. Case ii.

[5] Davis, C., "Case of Remarkable Pulsation in the Veins," *Dublin Hospital Reports*, 1827, vol. iv. p. 272.

[6] Benson, C., "Case of Pulsation of Veins," *Dublin Quart. Journ. of Med. Sc.* Nov. 1835.

[7] Elliotson, J., in his translation of Blumenbach's "Institutions of Physiology," London, 1820, p. 83.

[8] Beyer, A., "Observation sur une pulsation des veines superficielles," *Journal Complimentaire*, 1825, tome xxi. p. 330.

[9] Ward, T. O., "Venous Pulsations," *Lond. Med. Gaz.* 1832, vol. x. p. 376.

[10] Graves, R. J., "Observations on the Treatment of Various Diseases," *Dublin Quart. Journ. of Med. Sc.* Sept. 1834.

[11] Palmer, J. F., in his Edition of The Works of John Hunter, London, 1835, vol. iii. p. 227.

[12] *Comptes rendus*, 1858, p. 159.

[13] Quincke, H., "Ziemssen's Cyclopædia of the Practice of Medicine," vol. vi. p. 359.

[14] Broadbent, W. H., "Pulsatile Flushing, or Reddening of the Palms of the Hands and venous Pulsations," *Lancet*, 1875, vol. i. p. 466.

[15] Osler, W., "The Principles and Practice of Medicine," 1892, pp. 689 and 980.

health, as the personal observations of Wilkinson King,[1] Quincke,[2] and Hippesley[3] testify.

The beginning of a better appreciation of this hitherto confusing subject was made when first graphic records of the venous pulse were obtained, although, for a long time, it seemed but to introduce elements of greater discord. In the same year (1863) Bamberger[4] and Marey[5] published tracings of the venous pulse. Concerning the latter's tracing, which is not very distinct, its form rather inclines me to doubt Marey's interpretation of the curve. Bamberger's tracings appear to support his interpretation. They are practically single waved, and occurred in cases of undoubted tricuspid incompetence, and were caused by the ventricular systole. His observations were followed by those of Geigel[6] and Friedreich,[7] the latter entering very thoroughly into a discussion of the subject. Friedreich contested Bamberger's hypothesis that the venous pulse was a sign of tricuspid incompetence, and demonstrated its presence in a number of patients in whom there was no heart affection. One of the most characteristic forms that the venous pulse assumed in his tracings was that in which a small wave preceded the larger and chief wave (anadicrotic venous pulse). He assumed that this small wave was due to the auricular systole, and the larger wave to the ventricular systole. A careful study of his cases and of his tracings leaves no doubt that when he found this venous pulse in cases with no heart affection, he was misled by the similarity of these curves to those obtained from patients with serious heart disease. To account, then, for the occurrence of the venous pulse in people with healthy hearts, he assumed that during the ventricular systole the ventricle projected funnel-like into the auricle, and thus sent back a wave into the jugulars, so that all that could be inferred from such cases was an incompetence of the jugular valves. I shall show later that the wave which he assumed to be ventricular was in reality due to the contraction of the auricle. So far the tracings had been taken alone and independently of any method of timing them with some standard movement (heart beat or arterial pulse).

[1] *Loc. cit.* p. 108.

[2] Quincke, H., "Beobachtungen ueber Capillar-und Venenpuls," *Berlin. klin. Woch.* August 1886, No. 34, p. 357.

[3] Hippesley, J., "Pulsation in the Veins," *Nature*, London, 1885, vol. xxxii. p. 389.

[4] Bamberger, H., "Beobachtungen ueber den Venenpuls," *Würzburger med. Zeitschr.* 1863, Bd. iv. p. 232.

[5] Marey, E. J., "Physiologie médicale de la circulation du Sang," Paris, 1863, p. 531, fig. 216.

[6] Geigel, A., "Ueber den Venenpuls," *Würzburger med. Zeitschr.* 1863, Bd. iv. p. 332; and "Weitere Beobachtungen ueber Insufficienz der Tricuspidalis und Venenpuls," *ibid.* 1865, Bd. vi. Heft 5, p. 249.

[7] Friedreich, N., "Ueber den Venenpuls," *Deutsches Archiv f. klin. Med.* 1865, Bd. i. p. 242.

In 1868, Potain[1] obtained tracings of the venous pulse simultaneous with that of the carotid. The chief features in these tracings were (1) a wave preceding the carotid pulse, and therefore due to the auricular systole; and (2) a great depression during the auricular diastole. Other features are present, but, as I consider them misinterpreted, I will refer to them when dealing with the similar forms in my own tracings. Inasmuch as this pulse was present in presumably healthy people he looked upon it as the normal venous pulse, or the false venous pulse, in contradistinction to that form which occurs in advanced heart disease synchronous with and caused by the ventricular systole (the true venous pulse).

John Hunter,[2] in considering the factors that move the blood in the veins, incidentally refers to the fact that, with the expulsion of the ventricular contents from the chest, a vacuum would arise which would expedite the flow of blood into the auricle during the ventricular systole. Brücke[3] demonstrated experimentally what he considers the effects of this influence on the veins, and Mosso[4] following him obtained tracings undoubtedly due to this cardiac aspiration, from the movements of the air contained in the air passages. These movements, registered at the same time as the carotid pulse, present features of similarity to tracings of the pulsations in the veins, inasmuch as in both there was a great fall during the ventricular systole. He, therefore, assumed that the pulsation in the veins was essentially a negative pulse, in that the depression or negative wave merely represented the greater suction influence of the thorax at the time of the expulsion of the ventricular contents. This view was disproved by Gottwalt,[5] Riegel,[6] and François-Franck,[7] who found that this form of the venous pulse persists in animals when the chest is laid open, and when, in consequence, the supposed effects of the cardiac aspiration is abrogated. Although these authors undoubtedly disposed of Brücke's and Mosso's contention, they leave unexplained several features in Mosso's tracings, and as these are of some importance I shall have occasion to refer to them. Riegel and François-Franck followed up their researches on this subject both by experimental and clinical observations, and their views have to a certain extent displaced those of Friedreich in recent medical literature. Their conclusions are, shortly, that there is a normal venous pulse whose main features consist

[1] Potain, "Des Mouvements et des Bruits qui se passent dans les veines jugulaires," *Mémoires de la Société Médicale des Hôpitaux de Paris*, 1867, tome iv.

[2] Hunter, J., The Works of, Palmer's edition, 1835, vol. iii. p. 226.

[3] Brücke, E., "Vorlesungen ueber Physiologie," Wien, 1875, 2nd edition, p. 166.

[4] Mosso, A., "Die Diagnostik des Pulses," Leipzig, 1879, p. 46.

[5] Gottwalt, E., "Der normale Venenpuls," *Pflüger's Archiv*, vol. xxv. p. 23.

[6] Riegel, F., "Zur Kenntniss von dem Verhalten des Venensystems unter normalen und pathologischen Verhältnissen," *Berlin. klin. Woch.* 1881, No. 18, p. 249.

[7] François-Franck, "Part prépondérante du relâchment de l'oreillette droite dans le premier affaissement du pouls veineuse jugulaire," *Gazette Hebdomadaire de Médicine et Chirurgie*, 10th Feb. 1882, p. 92, *et seq.*; also *Compt. rend. des Séances et Mémoires de la Société de Biologie*, 1882, pp. 47 and 69.

of a great wave due to the auricular systole, a great depression due to the auricular diastole, and a secondary wave of variable importance about whose causation they give conflicting and very uncertain explanations. There is also a venous pulse, undoubtedly due to the ventricular systole, occurring only in advanced heart disease, and in time either systolic, or presystolic-systolic when some effect is still produced by the auricular systole. Frequent reference will have to be made to the works of these authors in the course of this paper, when their results and views are more fully discussed. Practically, however, this description is that generally adopted, except in rare cases where some other cause has been suggested as producing a venous pulse, *e.g.* Brunton's[1] description of a systolic pulsation in the left jugular vein due to the compression of the left innominate vein by the aorta.

The question whether the jugular pulse is a sign of tricuspid incompetence or not has been left in a very ill-defined state. Bamberger,[2] Riegel,[3] and others, state most positively that the only evidence is the systolic or presystolic-systolic pulse. Parrot[4] gives a series of cases where there was no valvular disease, but where there was supposed to be functional incompetence of the tricuspid valves, and he found support for his views in the fact of the presence of the venous pulse. While his views may be correct, there is little doubt that his observations on the time of the occurrence of the venous pulse are not reliable. Gibson,[5] who has written two luminous and instructive articles on this subject, gives a list of 80 cases in which the venous pulse occurred, and the diseases from which these patients suffered give a fair idea of the conditions predisposing to the functional form of incompetence of the tricuspid. But he does not distinguish the various forms of the venous pulse. Perhaps the state of the present knowledge (and confusion) relative to this subject is seen best in Peter's[6] work. According to this author the question of the venous pulse is a very simple one. There is a pulsation synchronous with the ventricular systole which is due to tricuspid incompetence—this is the true venous pulse. Then there is the pulsation presystolic, due to hypertrophy of the right auricle and not due to insufficiency of the tricuspid valves—this is the false venous pulse. Further, he describes the venous pulse of chlorosis, and agrees with Parrot

[1] Brunton, T. Lauder, "On Pulsation in the Jugular and other Veins," *Medical Press and Circular*, July 1879, p. 1.

[2] *Loc. cit.*

[3] Riegel, F., "Zur Diagnose der Tricuspidal-insufficienez," *Berlin. klin. Woch.* 1886, No. 38, p. 621.

[4] Parrot, "Étude clinique sur le siége et le mécanisme des murmures cardiaques dits anémiques," *Archives générales de médecine*, 1866, vol. ii. p. 129.

[5] Gibson, G. A., "Jugular Reflux and Tricuspid Regurgitation," *Edin. Med. Journ.* May 1880, vol. xxv. p. 979. Also "Action of the Auricles in Health and Disease," *ibid.* August 1882, vol. xxviii. p. 118.

[6] Peter, M., "Traité clinique et pratique des maladies du Cœur," etc., Paris, 1883, p. 620.

that there is in chlorosis a tricuspid incompetence, but of the form of the venous pulse, and of its connection with tricuspid incompetence, he is silent.[1]

2. *The Liver Pulse.*—In the works of the older writers no distinct mention is made of pulsation of the liver due to regurgitant waves of blood from the right heart; yet, from the study of numerous reported cases, we may readily infer that certain movements in the epigastrium and other parts of the abdomen, in cases of advanced heart disease, were in all probability genuine liver pulsations. Burns[2] appears to have been the first to recognise the possibility of the pulsation of the liver. Curiously enough he signifies that such pulsation would be due to contraction of the auricle, a cause which has been practically ignored by the great majority of observers, but one which, as I trust to show, is far from being uncommon. So far as I can see, however, Burns' statement was based more on hypothetical considerations than on practical observations. No very definite conception of this hepatic pulsation was attained until Friedreich[3] graphically demonstrated its occurrence in cases of tricuspid incompetence, and looked upon it as an earlier and more distinctive sign of this incompetence than the venous pulse. Following him, Mahot[4] published a series of cases, with tracings taken simultaneously with apex beat or arterial pulse, and these demonstrated the systolic occurrence of the venous pulse. The difficulty of recognising the liver pulse, and of distinguishing it from a variety of other movements (heart, aorta, vena cava, etc.), made writers extremely cautious in dealing with this subject. Several writers described a pulsation of the inferior vena cava, and Seidel[5] and Geigel cite cases of this supposed pulsation which Thamm[6] later, on experimental considerations, disputed. Mosso,[7] while protesting against adding confusion to an already confused subject, brought forward his view of the liver pulsation, suggesting that it was due to the "cardiac aspiration" already referred to. Although his views in regard to the venous pulse were disproved no one yet appears to have differentiated between the liver pulse and the results that Mosso obtained. As these have a very important bearing upon the matter of differential diagnosis, I will consider the subject at some length in speaking of the liver pulse. Gradually the possibility of its occurrence became more generally recognised, and cases were recorded by Thamm,[8] Taylor,[9]

[1] Peter, M., *loc. cit.* p. 626.

[2] *Loc. cit.* p. 625.

[3] *Loc. cit.*

[4] Mahot, M. F., "Des Battements du Foie dans l'insuffisance Tricuspide." Thèse. Paris, 1869.

[5] Seidel, M., "Pulsation der Vena Cava Inferior bei Insufficiencz der Tricuspidalis," *Deutsche Klinik*, 1865, No. ix. p. 81.

[6] Thamm, A., "Beiträge zur Lehre ueber Venenpuls und Gefässgeräusche, *Berlin. klin. Woch.* March 1869, p. 123.

[7] *Loc. cit.* p. 56.

[8] *Loc. cit.*

[9] Taylor, F., "On Pulsation of the Liver," *Guy's Hospital Reports*, 1875, vol. xx. p. 377.

Ramskill,[1] Drummond,[2] Riegel,[3] Fenwick,[4] Smith,[5] Sainsbury,[6] and others, while additional tracings have been published by Galabin,[7] Taylor,[8] Riegel,[9] and Sansom.[10] But as their tracings were taken without any standard of time, little reliance can be placed on the interpretations of the various recorded events. So far it appeared to be considered that the liver pulse was purely ventricular systolic in time—or presystolic-systolic according to Riegel. Moore [11] indeed describes having recognised a pulsation in the liver, post-systolic in time. Drummond [12] considered the liver pulse as the sum of two movements: one due to the action of the heart mechanically depressing the liver, while the other was due to a genuine distension of the liver. He records a case where there was a double pulsation to each heart beat, which I suspect may have been due to both the auricular and ventricular systoles in the manner I shall show. Limperopoulo [13] in his thesis details a large number of observations, but it is somewhat difficult to unravel the events recorded in some of his tracings, which are generally taken with the heart beat or arterial pulse. Most of his genuine liver pulsations are those synchronous with the ventricular systole, but two of his tracings (3 and 6) are from cases where he considered there was a liver pulse due to the auricular systole, but the tracings are very imperfect, and no satisfactory information can be derived from their study. The point of interest, however, is that he describes an auricular systolic impulse, a feature also recognised by Peter.[14]

While, then, the majority of observers recognise a liver pulse, they practically all attribute it to great tricuspid regurgitation, and associate it with the systolic or so-called true venous pulse. This association has been demonstrated by Geigel,[15] and later by Pasteur,[16] and myself,[17] in that by pressure on the inferior vena cava, or liver, a distension of the jugular

[1] Ramskill, "Two Cases of Pulsating Liver," *Brit. Med. Journ.* March 1876.

[2] Drummond, D., "On Pulsating Liver," *Dublin Quart. Journ. of Med. Sc.* 1881, vol. lxxii. pp. 289 and 412.

[3] Riegel, F., "Ueber den normalen und pathologischen Venenpuls," *Deutschcs Archiv f. klin. Med.* May 1882, Bd. xxxi. p. 1.

[4] Fenwick, B., "A Case of Tricuspid Stenosis," *Trans. Path. Soc. London*, 1883, vol. xxxiv. p. 35.

[5] Smith, W. G., "Case of Pulsating Liver," *Dublin Quart. Journ. of Med. Sc.* 1884, vol. lxxvii. p. 55.

[6] Sainsbury, H., *Lancet*, Nov. 1890.

[7] Galabin, A. L., "On the Construction and Use of a New Form of Cardiograph," *Medico-Chir. Trans.* 1875, vol. lviii. p. 353, fig. 9.

[8] Taylor, *loc. cit.* [9] Riegel, *loc. cit.*

[10] Sansom, A. E., "The Diagnosis of Diseases of the Heart," etc. London, 1892, p. 545, fig. 172.

[11] *Brit. Med. Journ.* 1884, vol. i. p. 168. [12] *Loc. cit.*

[13] Limperopoulo, A., "Les pulsations hépatiques dans l'insuffisance tricuspide. Thèse. Paris, 1891.

[14] *Loc. cit.* p. 622. [15] *Loc. cit. Würzburger med. Zeitschrift*, 1865, Bd. vi.

[16] Pasteur, W., "On a New Method of Estimating the Condition of the Right Side of the Heart," *Lancet*, 1886, vol. i. p. 914. Also *ibid.* Sept. 1885.

[17] *Loc. cit.* fig. 37.

veins ensued. Another form of liver pulse deserves a passing notice. Rosenbaeh[1] has described a pulsation in the liver due to the direct injection of the liver by the hepatic artery—an arterial pulsation. This form must be of exeessive rarity, though the possibility of its oecurrenee might be inferred from what is seen in the brain, and from the scanty reeords of pulsation in the spleen,[2] pneumonie lung,[3] or eerebriform tumours.[4] Probably to this form would belong Drummond's case of Graves' disease with pulsating liver. While I eannot speak of this form I would point out that the eireumstanees attendant upon its presenee are likewise those in whieh an impulse is transmitted to the liver by the emptying and filling of the ventrieles, with which I shall have to deal more fully hereafter.

Section II.—Methods.

Hitherto there have militated against the proper recognition of venous pulsations two factors, namely, the laek of a convenient method for reeording them, and a misconeeption of their nature, fostered by the applieation of question-begging names. In regard to the first, no satisfaetory elinieal method has been devised. A prime necessity in such an instrument is the ability to register, at one and the same time as the venous pulse, some known movement of the heart as a standard (apex beat or arterial pulse). The methods hitherto adopted by observers have been the employment of instruments, of whieh the main elements consisted either of a steel spring and writing lever,—the former applied direetly over the vein,—or air transmission by means of tambours, various devices being employed to make the pulsating vein eommunicate its movements to the tambour. It has been found praetieally impossible in the former ease to obtain at one and the same time movements of the venous and arterial pulses, but by means of air transmission methods this end is readily attained. Here, however, the neeessity for elaborate and bulky apparatus for reeording purposes has restricted the employment of the method to sueh narrow limits that the subjeet has only been investigated to a limited extent.

For the purpose of this inquiry I have employed three sets of instruments, and for the due appreeiation of the results obtained it will be neeessary to describe them in some detail.

1. *The Phlebograph.*—The essential parts of this instrument are—(1) a small cup for receiving the impressions of the pulsation, (2) a tube for trans-

[1] Rosenbaeh, O., "Ueber arterielle Leber Pulsation," *Deutsche med. Woch.* 1878, Nos. 40, 41, and 42.

[2] Gerhardt, C., "Aorteninsuffieienz und Milz Pulsationem," *Centralbl. f. klin. Medicin*, 1888, No. 1.

[3] Graves, R. J., "Clinieal Lectures on the Praetiee of Medieine," *New Syd. Soc.* vol. ii. p. 46.

[4] Stokes, W., "A Treatise on the Diagnosis and Treatment of Diseases of the Chest," *New Syd. Soc.* pp. 292 and 391.

mitting the impressions to (3) the tambour and lever, and (4) a recording apparatus. The small cup for receiving the impressions (which will hereafter be referred to as the "receiver") is simply a small shallow vessel, circular in shape, $1\frac{1}{4}$ in. in diameter, and $\frac{1}{2}$ in. in depth. The open mouth is applied over the pulsating part in such a manner that its edges are so closely adapted to the skin that no communication is kept up with the outer air. From the roof of the receiver rises a narrow pipe, $\frac{1}{2}$ in. in length. To this pipe is fitted an india-rubber tube from 3 to 4 ft. in length, the other end of which is connected with the tambour. The tambour is $1\frac{1}{4}$ in. in diameter and $1\frac{1}{2}$ in. in height. The object in making it so large is simply that the bottom of it might be made of solid metal, and thus give it sufficient weight to prevent its too free movement when placed upon a table. The lever attached to this tambour is jointed at its middle, the joint moving stiffly, so that it is maintained in any position in which it is placed. The object of this is to permit of the more ready adaptation of the lever joint to the recording apparatus at any desired height. The recording apparatus is simply a Dudgeon's clockwork sphygmograph, adapted to the purpose. The instrument is tied to its box, with the clockwork portion uppermost, and the blackened paper then moves from right to left in such a manner that, with a little manipulation, the lever of the tambour inscribes its movements thereon.

The site usually selected for taking a tracing of the venous pulse from the internal jugular vein is at the root of the neck on the right side, immediately above the clavicular attachment of the sterno-mastoid muscle. The patient being in the recumbent position, the head is generally placed with the chin pointing slightly upwards and to the left, in such a manner that there is not the slightest tension on the sterno-mastoid muscle. The receiver placed over the yielding muscle, or slightly behind it, receives the impulse very readily. There is frequently a fusiform dilatation of the internal jugular about the middle third of the neck, and when this pulsates excellent tracings can be got from the swelling. Sometimes, in advanced cases, the recumbent position causes such distension of the veins that the pulsation ceases. Then tracings may be obtained whilst the patient sits or stands up. Occasionally better tracings are got from the right internal jugular vein.

It frequently happens, particularly in fat people, that the yielding tissues fill up the cavity of the receiver to such an extent that the lever of the tambour is raised to an inconvenient height. This can sometimes be avoided by tilting up the sides of the receiver, and not closing it on the skin till the desired situation is obtained. It is still more satisfactory, however, to employ a tambour with a small air hole, and when the receiver is placed in the desired situation to close the air hole with the finger of the hand that holds it in position.

In taking tracings of the liver pulse a modification of this receiver is necessary. For such purposes the liver receiver should be larger. The one employed in these observations was 5 in. in length, 2 in. in breadth, and 1 in. in depth, its open edges were curved slightly on its long axis, so as to allow of its being readily adapted to the abdominal surface. Here an air hole is absolutely necessary. In employing the liver receiver, the position of the lower margin of the liver having been ascertained, the

receiver, held in the right hand, is laid lengthwise across the abdomen, its lower edge being 2 in. below the liver margin. Steady continuous pressure is applied to the lower margin of the receiver till it sinks deeply into the abdomen, and then the upper margin is adapted closely to the skin. In this manner a considerable space of the lower liver edge is embraced by the receiver. If the finger be now applied over the air hole, and if the receiver is in a suitable position, the movements of the respiration will be communicated to the lever. If the patient now ceases to breathe, the liver movements alone are transmitted. Having found that the receiver is in position, the lever is adjusted with the left hand to any desired point on the recording surface, the patient again holds the breath, and the clockwork being started, the movements of the lever are readily recorded. The receiver should be applied as far to the right as possible, in order that any impulse from the abdominal aorta may be avoided. It is advisable to take but a few liver pulsations at one time, as frequently the patient can only hold the breath for a brief period, the effort often producing a hardening of the muscles of the abdominal wall, which spoils the opportunity for obtaining a good tracing. Even in cases of considerable dyspnœa, the period of a few seconds that a patient holds the breath is generally sufficient to enable a very accurate tracing to be obtained. It sometimes happens that the abdominal parietes are so rigid that all attempts to obtain a liver tracing are unavailing.

Useful as I have found this instrument, it still left a good deal to be desired. It was convenient and could be easily carried about, but when it was desirable to make an accurate observation as to the time of the events I had to employ a cumbersome recording apparatus. In the majority of cases the features of the tracings were so evident that such timing of the events was unnecessary. Still it militated against the easy and rapid assurance that was necessary for the study of this subject.

For some time I had been making observations on the relations of the respiration to the pulse, and to facilitate these observations I had devised an arrangement by which a tambour with a short lever was attached to a Dudgeon's sphygmograph, the respiratory movements being conveyed to the tambour from an india-rubber bag strapped to the chest or abdomen. In this manner a simultaneous record of pulse and respiration was obtained (Fig. 5). It struck me that the instrument might be modified, so as to be utilised for recording the venous pulse at the same time as the pulse of the radial artery. For that purpose I had the following instrument constructed, which answers the purpose admirably.

2. *The Clinical Polygraph.*—Inasmuch as the whole arrangement can be used for taking, at the same time and on the same recording surface, tracings of the radial pulse, with tracings of the apex beat, carotid, venous, or liver pulse, or the respiratory movements, and as its size is such as to permit its

being carried about with the greatest facility, and readily employed in general practice, I will refer to it as the "clinical polygraph."

The instrument consists of a tambour (Fig. 1, *A*), supporting the writing lever, the latter being 6 in. in length. From the under-surface of the tambour a pipe protrudes, which is connected by the india-rubber tubing with a receiver. Screwed tightly to the bottom of the tambour is a stem *B*, 6½ in. in length, projecting outward, parallel to the under surface of the tambour. Half an inch of the other extremity of the stem is bent almost at right angles *C*, and this portion fits into a slot in the upright stem that supports the movable lever of a Dudgeon's sphygmograph (Fig. 2), and this supports the tambour. When the tambour arrangement is adjusted to the sphygmograph, the tambour rests with its movable surface vertically, so that the writing lever moves horizontally. For the purpose of adjusting the point of the writing lever to any desired place on the recording paper the following movements are available. The point of the lever can be approximated to, or withdrawn from, any desired point by means of a sliding arrangement at *D*, where the stem consists of two parts clasping one another. The writing point can be made to move in the horizontal direction, so as to write at any desired level on the paper, by means of a joint at *E*, which moves stiffly, and retains the position in which it is placed. Finally, the lever can be raised out of reach during the adjustment of the sphygmograph to the pulse, and brought back and allowed to touch the surface of the blackened paper with sufficient delicacy to permit its movements to be accurately recorded, but not restrained by too close pressure, by rotating the tambour at a joint formed where the stem is fixed to the under-surface of the tambour. By means of these arrangements the lever can be moved with great facility and accuracy in a vertical and a horizontal direction, as well as made to approach or recede from any given point.

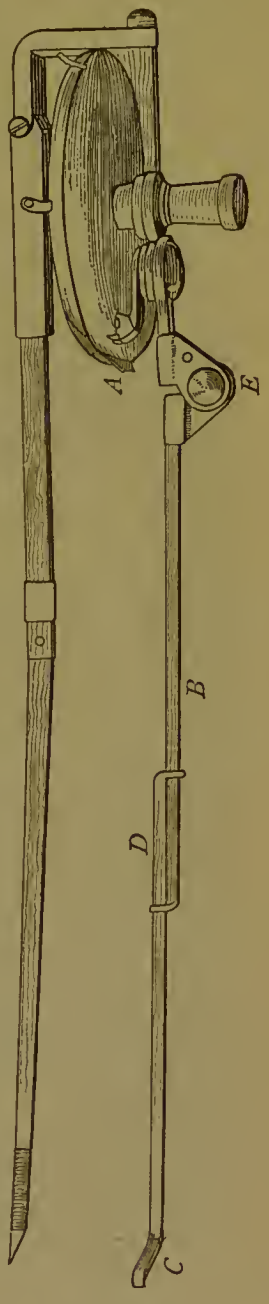

Fig. 1.

By this method any movement can be recorded at the same time, and on the same paper as the radial sphygmogram. One can make the tambour lever write directly above or below the writing lever of the sphygmograph, or, when the movements are large, a little behind, so that the two levers do not come into contact during their excursions. Ordinates are obtained by allowing the lever to make distinct marks on the paper before starting and after stopping. With a pair of compasses the relative time of the events can be accurately gauged.

Some difficulty may be experienced in the employment of the clinical polygraph, on account of the weight of the tambour tilting the sphygmograph off the radial pulse. If we employ the inelastic band usually supplied with the sphygmograph, this inconvenience may possibly be very serious. I have long ago abandoned the use of the inelastic band (placing no reliance on the pressure supposed to be required to obtain an idea of the arterial tension), and employ instead an elastic band, tying the instrument to the wrist by a knot. Should any slipping occur, the sphygmograph and tambour can readily be readjusted. It frequently happens that the radial pulse causes such a large excursion that there is no clear space on

the paper for the venous or other pulsation. In such a case I either shift the sphygmograph till I obtain a tracing of less amplitude (seeing that the object of taking the radial pulse at the same time as the venous is to obtain an

Fig 2.—Taken from a photograph, showing the method for taking a liver tracing and a radial tracing by means of the clinical polygraph. In the process of enlarging the original, the position is reversed and made to appear as if the tracing was being obtained from the left side of the patient. As, however, the object of the engraving is to show the clinical polygraph, the illustration does that in a satisfactory manner.

idea of the relative time), and, before the paper has entirely passed through, stop the clockwork, remove the tambour, and obtain a full-sized tracing of the radial pulse (Fig. 3).[1] Or the venous pulse may be taken a little behind the radial, and the various events disentangled. For accurately estimating

[1] All the tracings read from left to right.

the time of the events it is advisable to take on the same paper a few beats of the carotid pulse. For convenience of study more events can readily be

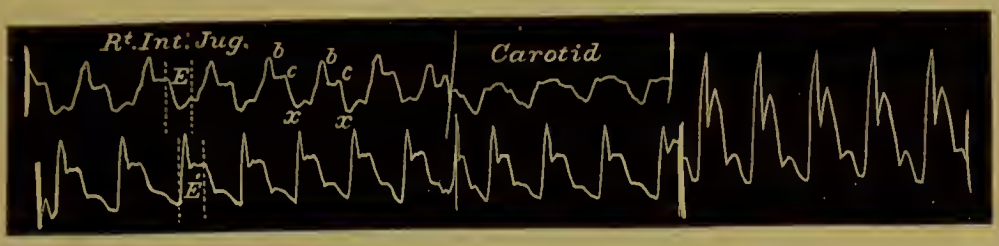

FIG. 3.—The first part of the tracing shows the venous pulse from the right internal jugular vein (upper tracing) taken at the same time as the radial pulse (lower tracing). The clockwork being stopped, the receiver was applied over the carotid, and a tracing of the carotid and radial taken. Again the clockwork was stopped, and the tambour portion of the polygraph removed, and the full effect of the radial pulse was inscribed on the last portion.

recorded within the limits of a short sphygmographic paper; and a record of apex beat, venous, carotid, and liver pulses may be taken at the same time as the radial sphygmogram (Figs. 3 and 4). When a long tracing is required

FIG. 4.—Apex beat, carotid, right internal jugular, and liver pulse (upper tracing), taken at the same time as the radial pulse (lower tracing).

(as for instance in noting the movements of the two sides of the heart in cases of occasional irregularity), a tracing paper 1 or 2 ft. in length may be employed (Figs. 98 and 101).

This instrument is also capable of registering the respiratory movements at the same time as the radial pulse. The receiver is placed over the episternal notch, or above the clavicle, the inspiration then causes a depression in the tracing (Fig. 5). Or a bag tied round the chest or abdomen may be used. Inspiration then causes an upward movement in the tracing. The size of the

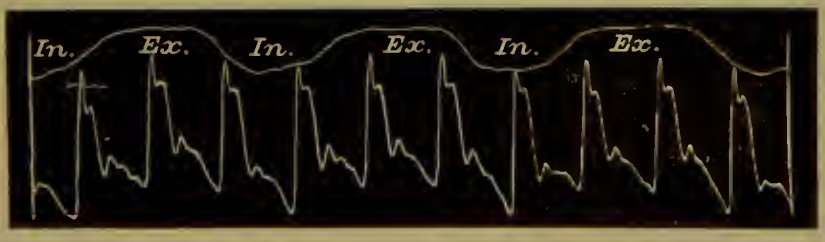

FIG. 5.—Simultaneous tracings of the respiratory movements and the radial pulse, from a case of bronchitis with laboured breathing.

clinical polygraph is such that it can be readily carried in the pocket. A case containing a compartment for the tambour portion, and space for twelve ordinary-sized papers, measures $6\frac{1}{2}$ in. × 3 in. × 1 in.[1]

[1] The apparatus has been made for me by Messrs. Krohne and Sesemann of London.

3. *Knoll's Polygraph.*—The greater number of my observations have so far been made by means of a Knoll's polygraph.[1] This instrument consists essentially of a cylinder 4 in. in diameter, resting on the clockwork frame. Two tambours are fixed, the one above the other, to an upright stem, at such a distance that the points of their levers touch the cylinder. The receivers already described are adjusted to the india-rubber tubing connected with the tambours. The levers can be made to write the one directly under the other, so that an opportunity of noting the time of coincident events is readily afforded.

The methods thus employed are open to some objections. In the case of the clinical polygraph, a comparison is made between movements recorded by different instruments. But in the deductions drawn from the results no attempt is made to compare the character, but only the time of the various curves. It is assumed that the time occupied by the pulse wave in the veins will take the same time to travel from the heart to the jugulars as the arterial wave from the heart to the carotids. Possibly the difference in the structure of the walls, and the resistance offered to the wave, may make a difference in the rate of propagation, but if so it is so slight that it need not be considered. In employing the radial pulse to time the events of the jugular pulse, a distinct loss of time must be allowed for on account of the radial pulse appearing later than that of the carotid. But this difference is readily estimated by taking, immediately after the venous pulse has been recorded, a tracing of a few beats of the carotid pulse, and in calculating the time of the different events in the jugular pulse by the standard of the radial pulse, allow for the difference between the carotid and radial. It will be found that very little time is lost by air transmission, as compared with the lapse of time between the carotid and radial pulses. In Fig. 6 the tracing of the venous pulse from the right jugular was taken by means of the direct application of a steel spring with attached lever, at the same time that the pulse from the left jugular was taken by the air transmission method. It will be found that there is very little difference in time; whereas the time of the carotid pulse, conveyed by this method to the sphygmograph on the radial artery, shows the carotid pulse appearing usually at a distinct and appreciable interval before the radial pulse.

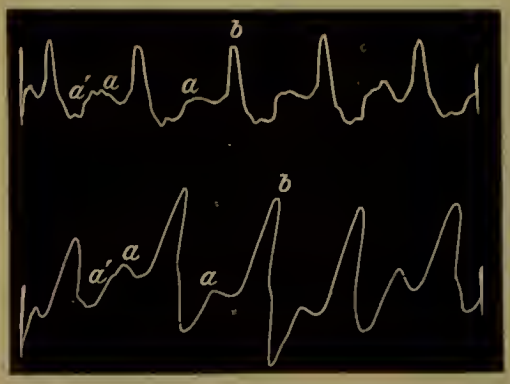

Fig. 6.—Simultaneous tracings from the right jugular bulb (lower tracing), by means of a steel spring and lever, and from the left jugular bulb (upper tracing), by means of the receiver and air transmission method (Case 2).

Another objection can also be raised against the air transmission

[1] For illustration of polygraph, see *Journal of Pathology and Bacteriology*, 1892, vol. i. p. 88.

method:—the tendency to inertia vibrations. No doubt this might be a serious objection; but similar objections can be urged with more or less force against all graphic methods. Even if an imperfect instrument is employed, and its imperfections are recognised, and these movements produced by a faulty instrument are not mistaken for those produced by the pulse itself, much information can still be acquired. I have taken many precautionary measures to ensure accuracy of interpretation, both by careful inspection and by other methods. I have endeavoured to check the movements obtained by the air transmission method, by taking a series of tracings directly from pulsating parts by means of a steel spring. For this purpose I removed the tambour from a Knoll's cardiograph, and employed the steel spring which projects, tongue-like, from the concavity of the horse-shoe shaped rest. I attached a lever to the projecting stem, which is connected with the button at the free end of the spring. The button laid over the vein or apex beat readily communicated its movements to the lever, which inscribed them on the revolving cylinder. Practically the same movements were recorded in all cases, the tambour method occasionally showing small vibrations at the end of a rapid movement. In Fig. 7 are shown three cardiograms, taken by the steel spring (I), by a Knoll's cardiograph (II), and by the receiver (III). While there are present a few smaller differences, yet the general contour in all three cases is preserved. I have carefully avoided placing any interpretation upon tracings, even when one might have justifiably done so, when there was any possibility of these vibrations confusing the results obtained.

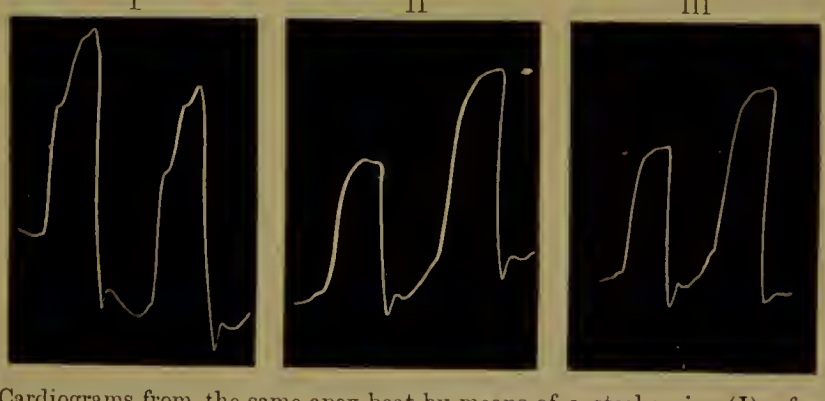

FIG. 7.—Cardiograms from the same apex beat by means of a steel spring (I), of a Knoll's cardiograph (II), and of the receiver (III).

The clinical polygraph is a very delicate instrument, inasmuch as the tambour, being placed sideways, all vibrations due to the weight of the lever being accelerated by gravity are excluded.

SECTION III.—THE TIMING OF THE EVENTS IN THE LIVER AND VENOUS PULSES.

As I have said, I have assumed that movements occurring in the jugular veins would be transmitted at the same rate from the heart as

are movements in the carotid. Therefore the carotid pulse forms the best standard. Sometimes, however, it is not available, either on account of the smallness of the arterial pulse, or because the size of the venous pulse renders the tracing imperfect. Usually I place the receiver over the external carotid immediately after it has branched off, as here it is superficial, and a venous pulse rarely extends so far. Except for the commencement of the arterial wave, the carotid is rarely available for any other period. The dicrotic notch is not always apparent, and it often happens that the elevation of the lever is maintained at its highest for a distinctly longer period than the ventricular outflow. The cause of this is that the lever follows the pulse wave only to a limited extent, and the highest points of the wave and the dicrotic notch are not represented. This might give the idea that there may be a want of reliability of the method in other respects, and might be regarded as a serious objection. But the relative accessibility of the pulsations must be borne in mind. The venous pulse is usually more accessible, and the receiver more readily adapted to its contour, whilst, also, the movements are less powerful and not so abrupt.

When the carotid has not been available, I have frequently employed the apex beat. I have made only limited attempts here to base any observations on the events in the apex beat tracings. I do not pretend to have obtained accurate apex beat tracings in all cases. So much discordance exists in regard to what constitutes a good cardiogram, and to the interpretation of the cardiogram, that it would be introducing, needlessly, a disputable subject were I to base any observation on the apex beat curves. I therefore employed the apex beat merely to indicate roughly the time and duration of the ventricular systole, and to note that the ventricular outflow must lie between the ascending and descending lines of the curves (Fig. 8). The time of the events taken with the apex beat as a standard have been rarely employed alone; but, when taken, due allowance has been made for the difference of time between it and the carotid pulse. When the tracings here give the apex beat as the standard, almost all have been verified in other ways.

In obtaining an accurate record of the time of the different events, it will be found that frequently a line is made upon the two tracings, indicating the coincidence of the events at that period of time. These ordinates are obtained in the following ways:—When the larger apparatus with the revolving cylinder is used, before the cylinder begins to revolve, distinct lines are made by making the levers move on the paper when the cylinder is at rest. When the cylinder revolves, these ordinates are obtained either by stopping the cylinder at short distances, or, more usually, by allowing it to complete a revolution and, after stopping, to make distinct lines at the end of each tracing. The cylinder is then rotated backwards, and stopped at desired intervals, and the marks made at these points by the two

levers. If the levers are maintained in the same position, they can only touch the identical points they would occupy during the revolution of the cylinder. Finally, they are allowed to fall on the ordinates made at the beginning of the tracing, to guarantee that they are still maintained in their relative positions. At other times, and particularly in tracings obtained by the polygraph, the periods marked were obtained by careful compass measurements.

In most of the tracings these marks show the period of time at which the event occurred; and, to find the relative time, it must be remembered that the apex beat precedes, and the radial pulse follows, events produced in the carotid arteries or jugular veins. Sufficient data will be found in most of the tracings for checking the interpretation of the events.

The time of ventricular outflow has occasionally to be referred to, and it is represented on venous and liver tracings by the space *E;* while, in the apex beat or arterial tracings it is represented by the space *É.* As an important part of the argument is based upon the time occupied by the ventricular outflow, I will here explain the method employed to obtain its duration. As a rule, I have based it upon the sphygmogram obtained from the radial pulse, and calculated the duration as lasting from the beginning of the upstroke to the fall immediately preceding the dicrotic notch. I have fixed upon this latter period, on the ground that, as the closure of the aortic valves produces the dicrotic notch, the time of the occurrence of the cause will precede that of the event; and just as we estimate the duration of the ventricular outflow from the beginning of the rise of the pulse wave, so in the converse condition, the time of the closure of the aortic valve should be estimated from the beginning of the fall. Frequently the line to the dicrotic notch is a slanting one, and I then estimate the time of closure a little before the lowest part of the aortic notch is reached.

I should have preferred to have given more reliable data for the time of closure of the aortic valves. The time assumed here is practically that given by Roy and Adami, and von Frey. Others, as Marey, Hürthle, and Porter, place the time at the bottom of the aortic notch; but a critical analysis of their evidence demonstrates the fact that their tracings do not bear out their contentions. Evidence based on the second sound is far from conclusive, first, because there is no proof that the mere closure of the valves produces the second sound (although, no doubt, the valves are otherwise associated with the production of the sound); and, second, the methods for obtaining a register of the sound (by auscultation and signalling, or microphonic-electrical stimulation of a frog's muscle—Hürthle) are liable to grave errors. If it be considered that the arguments advanced in this paper establish the time of closure of the pulmonary valves, it will be found that the time of the closure of the aortic valves adopted here agrees exactly with the time of

closure of the pulmonary valves. It is to be noted, however, that while the aortic-valve closure is coincident with a fall in the arterial wave, the pulmonary valve closure is coincident with a rise in the venous pulse. That this should be the case will be realised when the different ways in which the arterial and venous waves are affected by the closure of the aortic and pulmonary valves are considered. At the time of the closure of the aortic valves, there is a cessation of the ventricular flow into the artery, while, with the closure of the pulmonary valves, the outflow into the veins is accelerated, in the manner hereafter demonstrated.

As the spaces *E* and *É* represent the duration of the ventricular outflow in the several tracings, they will not occur at the same period of time as in the jugular pulse, except in the carotid, but will occur a little earlier in the apex beat tracings, and a little later in the radial pulse tracings.

Sometimes, when the sphygmographic tracing did not form the basis of the calculation of this period, I have used the apex beat, and have simply included the time between the ascending and descending lines, as the time of ventricular outflow (Fig. 8).

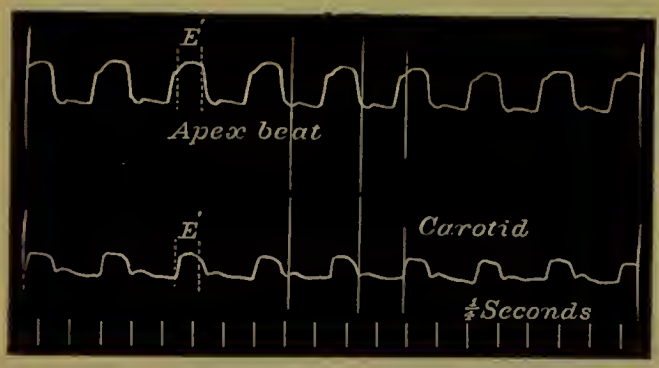

FIG. 8.—Simultaneous tracings of apex beat (upper tracing) and carotid pulse (lower tracing), with the time register in quarter seconds. The spaces *E* represent the duration of the ventricular outflow in the apex beat and the carotid pulse.

I may here explain, with regard to certain imperfections, both in carotid pulse and apex beat tracings, which may be detected in the tracings, that these were frequently obtained under somewhat disadvantageous circumstances. Thus, most of the observations have been made on patients in their own homes, and with no assistance except what the patient could afford. While I held the receiver over the venous pulse with one hand, I had to manipulate the levers and the instrument with the other. The patient then had to hold the receiver or cardiograph to the carotid or apex beat; naturally, under these conditions, I could not expect perfect tracings. I have not used any of the tracings where any doubt existed as to the interpretation, without employing various devices to guarantee the accuracy of the deductions. Since I have devised the clinical polygraph, I have been able to obtain, with great facility, more satisfactory results, as the patient's or other help is dispensed with. I have, however, in numerous instances, employed both the clinical and Knoll's polygraph in order to guard against any error.

Section IV.—The Effects of Respiration in the Venous Current.

Before entering upon the consideration of the tracings of the venous and liver pulsations, it will be advisable to consider two factors, whose presence has, occasionally, important effects upon the tracings; these are the influence of the respiratory movements and that of the carotid pulse.

Since the experiments of Carson [1] and Barry,[2] demonstrating the influence of the low intra-thoracic pressure of inspiration on the flow of blood in the veins (although Haller had previously pointed out this influence), no doubt has been entertained of the effects of inspiration on the flow of blood to the chest. From more recent experiments, however, it is shown that the flow, though less rapid, is still maintained during expiration.[3] In health there should be no visible stasis of the blood; but, as will be seen, the respiratory movements have a distinct influence on the curves produced by the pulsation of the veins. When, however, a distinct pulsation, due to the movements of respiration, occurs in the veins, there is some morbid change increasing the pressure within the thoracic cavity. Hamernjk asserted that this pulsation, synchronous with the respiratory movements, may be due either to simple stasis in the veins, during expiration, when the jugular valves were competent, or to the actual expulsion of blood into them, if the valves were incompetent. He further asserted that the jugular valves closed during expiration in health; but, as Skoda has pointed out, this is impossible, for the mere temporary closure with the finger produces a swelling, and naturally such a swelling would show itself if the valves closed during expiration in health. Immerman,[4] who made a special study of the subject, came to the conclusion that this swelling or pulsation due to respiration in the veins was an evidence of increased intra-thoracic pressure, due either to diminished elasticity of the lungs, or to obstruction to the entrance of air into the lungs, a variety of causes, which we need not consider here, being capable of producing this diminished elasticity and obstruction. The effects of the respiratory movements in modifying the venous pulse have to be reckoned with, and this tendency to dilatation in the veins during expiration will be occasionally demonstrated; the combined influence of respiration and cardiac regurgitation will, afterwards, be more fully considered.

Section V.—The Relation of the Arteries to the Veins.

With very few exceptions, all the tracings of the venous pulse that are here given were obtained from the internal jugular vein. As

[1] Carson, James, "Essays, Philosophical and Practical." Liverpool, 1822.

[2] Barry, David, "Experimental Researches on the Influence exercised by Atmospheric Pressure upon the Progression of the Blood in the Veins," etc. London, 1826.

[3] François-Franck, *Gazette Hebdomadaire*, March 3, 1882, p. 132.

[4] Immerman, H., "Zur Pathogenese und Ætiologie der sichtbaren exspiratorischen Schwellung der Halsvenen." *Deutsches Archiv f. klin. Med.* 1866, p. 327.

the method employed could not be trusted to record movements in the vein alone, but also frequently showed curves that were undoubtedly due to the carotid pulse, it is necessary to consider shortly the relation of the vein to the artery, and to demonstrate how the artery makes itself appear in the tracing. This is all the more necessary, as the failure to recognise this arterial influence has misled various observers. The intimate association of the arteries with their accompanying veins, has been long recognised; and Hunter,[1] Richerand,[2] and King[3] considered that arterial pulsation was one of the factors that caused the blood in the veins to move, this effect being favoured, as Ozanam[4] demonstrated, by the inclusion of arteries and veins in a common sheath. The sudden movement communicated to the surrounding tissues by the carotid pulse may be considered to act in two ways. While the artery expands in all directions, it of course pushes against the structures in its immediate neighbourhood. In doing so, it will elevate the tissues that lie superficial to it, and these being displaced will drag upon those more distant from the artery. It will thus happen that there are practically two influences of the carotid impulse—one of elevation and the other of depression. If the receiver be placed over the artery, a tracing of the arterial pulse will be obtained; if the receiver be placed a little distance away, then a depression occurs coincident with the arterial pulse (Fig. 9).

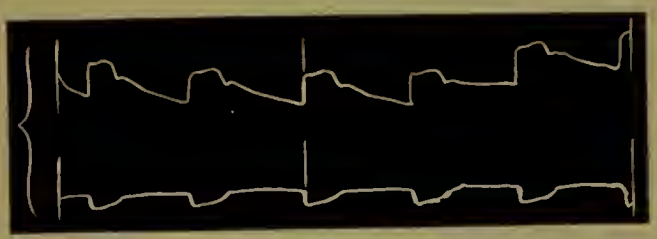

Fig. 9.—The upper tracing was taken with the receiver over the carotid artery, at the same time as the lower one was taken with the receiver about half an inch away from the carotid artery. The lower tracing is the inverse of the upper.

The lower tracing in Fig. 9 was taken half an inch behind the carotid in the middle of the neck, and shows a tracing the reverse of the upper taken from over the carotid—a negative carotid sphygmogram in fact. The patient from whom the tracing was taken (Case 32) had a small enlargement of the right thyroid gland, which pushed the carotid back, so that it lay alongside the posterior border of the sterno-mastoid muscle, and the neck was a very spare one. I mention this negative pulse wave, as a similar negative wave in the veins has been ascribed to this cause by Ozanam, and it will remove a possible source of misunderstanding, when the events in the venous pulsations are considered.

Arterial wave.—By the methods I have employed to obtain tracings

[1] Hunter, J., *loc. cit.* p. 227.

[2] Richerand, A., "Elements of Physiology," edited by J. Copland, 2nd edition, 1829, p. 186.

[3] King, W., *loc. cit.* p. 104.

[4] "Nouveau Dictionnaire de Médicine et de Chirurgie Pratiques," tome xxxviii. p. 649.

of the venous pulse, there are present in most of those where the pulsation is mainly diastolic in time, three waves. It will be convenient here to discuss the cause of the wave, which I have called an arterial wave, marked *c* in all the tracings.

I do not consider that this wave is produced in the vein at all, but look upon it as being solely the result of the carotid pulse. The jugular vein lies so close to the carotid that it is generally impossible to apply the receiver in such a manner as to avoid receiving also the carotid impulse. The reasons for assuming it to be purely arterial are as follows:—(1) It is always synchronous with the carotid pulse; (2) in moving the receiver away from the vein towards the artery, this wave assumes more and more the character of the arterial wave, or, in the course of recovery, the venous element may diminish or disappear, while the arterial element remains; (3) it bears a distinct relation to the character of the arterial pulse—large and well marked in cases of strong arterial impulse, faint and even imperceptible in cases of small and weak arterial impulse; (4) it is never present in the liver pulse, when, otherwise, the liver pulse bears a distinct resemblance to the venous pulse in character and rhythm; (5) during irregular heart action, when there is a pause in the arterial pulse, while the venous pulse continues, the arterial wave is absent from the venous tracing during this irregularity (Fig. 126).

Concerning the first of these reasons, many of the tracings to be given will amply bear this statement out, but I would call particular attention to Fig. 24, where the time of the occurrence of the arterial wave is found to be exactly the same as that of the carotid, even when the waves are widely separated by the rapid rotation of the cylinder. The second reason is exemplified in the same tracing: where the patient had, during expiration, a well-marked venous pulse; while, during inspiration, the arterial element alone remained (*C*). It will be seen that

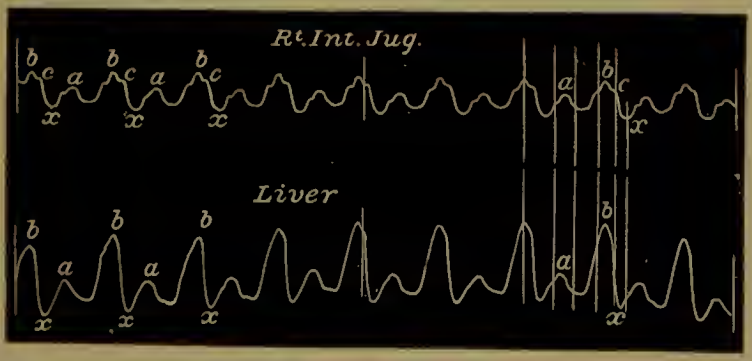

FIG. 10.—Simultaneous tracings of pulsations in the right internal jugular vein (upper tracing), and in the liver (lower tracing), to show the absence of the arterial wave *c* from the liver pulse (Case 8).

the wave *c*, at first a small wave with the venous pulse, comes to assume the character of an arterial tracing *C*. The marked character of the

arterial wave is seen in Fig. 45, when the carotid pulse was large; but in Fig. 15, where it was small, the arterial wave does not appear. When dealing with the liver pulse, it will be demonstrated that the features characteristic of that pulse are practically identical in rhythm,

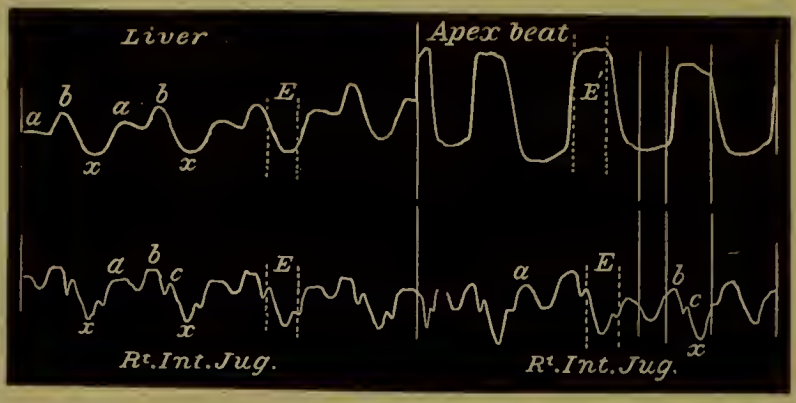

FIG. 11.—Simultaneous tracings of the liver and jugular pulses in the first portion, and of the apex beat and jugular pulse in the latter portion, to show the absence of the arterial wave *c* from the liver pulse (Case 46).

form, and origin, with those of the venous pulse. Figs. 10 and 11 show the venous pulse, with a well-marked arterial wave in both cases, taken synchronously with the liver pulse, and in the latter there is no sign of the arterial wave. That the wave is not produced by any defect in the instrument employed will be understood when it is found in tracings by other methods, as in Fig. 12.

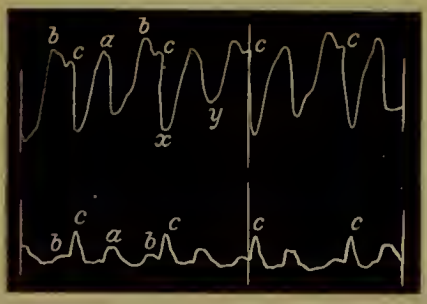

FIG. 12.—Simultaneous tracings of the pulse in the left jugular vein (lower tracing), by means of the receiver and tambour, and of the pulse in the right jugular vein (upper tracing), taken by means of the steel spring applied directly over the vein, to show the presence of the arterial wave *c* in the tracing taken by the direct method.

The arterial wave is to be found in the tracings taken by several observers. In Friedreich's tracings of the venous pulse from the normal heart,[1] it is frequently present, but as he misinterpreted the time of the event in these particular cases, his explanation is distinctly erroneous. In Potain's[2] tracing it is likewise distinctly visible, and is accounted for by him as a wave in the vein produced by the ventricular contraction. But, as will be pointed out, the appearance of such a wave from such a cause at this period is a physical impossibility. In some of Riegel's tracings it is also very evident. But when it appears here it has a

[1] Friedreich, N., "Krankheiten des Herzens," 2nd edition, p. 53, Erlangen, 1867; and "Ueber den Venenpuls," p. 24, figs. 26, 27, 28, and 29, *Deutsches Archiv f. klin. Med.* 1865, Bd. i. Heft 3.

[2] Potain, "Des mouvements et des bruits qui se passe dans les veines jugulaires," *Mémoires de la Société médicale des Hôpitaux de Paris*, 1867, p. 3.

rather deceptive appearance. Later, I shall have to dwell upon the changes that take place in the character of the tracing during rapid action of the heart, and it may, for the present, suffice to say that frequently only two waves appear—one produced by the auricle separated by a slight depression from that produced by the carotid arterial wave, as in Figs. 83 and 84. Curves somewhat similar to these are given by Riegel,[1] and I am more than suspicious that the tracings in his Figs. 8 and 9 are of a similar nature. I am all the more suspicious that this is the case when he attributes the latter of the two waves in his Fig. 8 to the closure of the tricuspid valve, and in his Fig 9 the latter of the two waves as due to the auricle, its contraction being delayed on account of the low tension. In numerous observations on similar curves I have established, beyond the possibility of doubt, the exact synchronism of this wave with that of the carotid pulse. In Mosso's[2] tracings this wave is to be detected, and he likewise points out its exact synchronism with the carotid pulse, but attributes it to the distension of the jugular vein prior to the suction exercised by the sudden diminution of the heart's size after expulsion of its contents. Pramberger[3] gives a tracing with this wave occurring during the auricular diastole; he considers that it is propagated from the aorta. The wave is to be recognised in a tracing given by Galabin,[4] but he does not attempt to account for its presence, and his interpretation of the whole tracing, which Sansom[5] adopts, is, in my opinion, incorrect.

Section VI.—The Relation of the Auricular Systole to the Venous Current.

The various factors that cause the blood to move in the veins, though intermittent in their action, produce practically a steady stream through the greater extent of the venous system. At the two extremities, however, there is a tendency, slight in health and exaggerated in certain forms of disease, to interference with this steady flow. The impulse of the heart may still impress intermittency through the capillary circulation, and be manifested in the smaller peripheral veins, more particularly when there is great dilatation of the arterioles, and hypertrophy of the heart, as in incompetence of the aortic valves. But this pulsation is distinctly a forward pulsation, and is distinguished chiefly from the

[1] Riegel, F., "Ueber den normalen und pathologischen Venenpuls," *Deutschcs Archiv f. klin. Med.* Bd. xxxi. Mai 30, 1882.

[2] *Loc. cit.* fig. 15, p. 64.

[3] Pramberger, H., "Ein Beitrag zur Beobachtung des normalen Venenpulses," *Wiener med. Woch.* 1885, Nos. 12 and 13.

[4] Galabin, A. L., "On the Construction and Use of a New Form of Cardiograph," *Medico-Chir. Trans.* 1875, vol. lviii. p. 353, fig. 4.

[5] Sansom, A. E., "The Diagnosis of Diseases of the Heart and Thoracic Aorta," fig. 171. London, 1892.

fact that its presence is limited to the peripheral veins, and is dependent on the arterial pulse, whilst the pulsation from the right heart is essentially a backward movement, and is most marked in the larger veins near the heart. It will not be necessary to refer further to the former of these pulsations, all the remarks hereafter having reference only to the retrograde movement communicated by the right side of the heart.

When the steady stream of venous blood approaches the heart it is subjected to the intermittent influence of the respiratory and cardiac movements. The former of these has already been shortly referred to, and it is now intended to enter more fully into the consideration of the latter. The powerful movements of the right ventricle, if communicating directly with the venous system, would exercise a deleterious influence upon the circulation. In health the effects of the ventricular movements on the venous circulation are prevented by the interposition of the auricle, whose chief function, as Fiek[1] expresses it, is to retain the pressure in the veins nearly constant. Nevertheless the movements of the auricle, even in health, appear to be communicated to the veins near the heart, though doubtless the amount of regurgitation is further limited by the peristaltic contraction of the ostial veins preceding the auricular systole. This movement is best observed in the valveless inferior vena cava (Haller, Bertin, Richerand) and its tributaries, when, according to Diemer[2] and Riegel,[3] it is constantly present, extending sometimes to the renal veins, and according to Bernard[4] and Diemer also to the liver veins. These movements are so slight as to be entirely beyond the reach of observation, except by experimental observations. Gottwalt, Riegel, and François-Franck, as already stated, have demonstrated their presence in the superior vena cava, and, to a limited extent, in its tributaries. Chauveau and Faivre[5] state that while a slight reflux occurs naturally during the auricular systole, it is much increased in the horse when the respiration is embarrassed. But it is doubtful if these pulsations are ever of sufficient strength in the healthy human body as to be visible. They are, however, of importance, as the time of their occurrence and exaggerated characteristics agree with the movements that occur most frequently as evident pulsations in the veins. The movements communicated by the heart to the veins are usually best observed in the internal jugular veins. The direct communication of the right internal jugular through the right innominate and superior vena cava in

[1] Fiek, A., "Der Kreislauf des Blutes," p. 27. Berlin, 1872.

[2] Diemer, L., "Ueber die Pulsationem der vena cava Inferior," Inaug. Diss. p. 13. Bonn, 1876.

[3] Riegel, F., "Ueber den normalen und pathologischen Venenpuls," *Deutsches Archiv f. klin. Med.* Bd. xxxi. Mai 30, 1882.

[4] *London Medical Record*, Oct. 15, 1878.

[5] Chauveau, A., and Faivre, J., "Nouvelles recherches expérimentales sur les mouvements et les bruits normaux du cœur," etc., *Gazette Médicale de Paris*, 1856, tome v. p. 406.

almost a straight line renders it readily sensible to the movements from the right heart. Its anatomical relation, and relatively superficial situation, render it also readily accessible for observation.[1] At a varying distance from ½ to 2 in. above the junction of the jugular veins with the subclavian a couple of valves are situated. Considerable doubt exists as to the competency of these valves. Reid[2] and Riegel[3] found by experimental observation that they were naturally incompetent, while François-Franck[4] came to an opposite conclusion. Clinical observers usually assume their competence, and even detailed observations are on record of a "jugular valve sound" during their forcible closure, which, when incompetency occurs, is replaced by a "jugular valve murmur." The sound is always stated to be ventricular systolic in time, whereas the time that the valves would close in the great majority of cases is presystolic in time, that is, during the auricular systole, as in Case 2. The test usually recommended to distinguish incompetence of the jugular valves is to compress the vein in the middle of the neck and observe whether the pulsation continues below the compressed part. This is far from being satisfactory, and besides is not of much consequence, if it be a fact, which I am inclined to believe with Bamberger[5] and Riegel, that the jugular valve usually readily becomes incompetent. When a pulsation is visible in the neck I am doubtful if mere stasis is capable of giving such a manifest movement of such force as to enable one to obtain a graphic record. The reason for this statement is based upon such facts as the following:— (1) I have on several occasions observed cases where the jugular valves were evidently competent, and the reflux from the heart was so powerful as to dilate the bulbous cavity at the junction of the jugular and subclavian veins into a distinct swelling. The movements of this swelling were very powerful, yet in some cases (even where the neck was thin) no pulsation whatever was visible in the veins during the pulsations in the bulb. At this period the valves must have been closed, and temporary stasis must have occurred in the veins. (2) In other cases a faint pulsation was observed in the jugular veins of the neck, but not sufficient to enable me to obtain a tracing. (3) During the administration of chloroform, when the patient strains and the veins become turgid, I have observed the valves of the external jugular become incompetent

[1] For a detailed account of the anatomical relations of the veins and of the aponeurosis covering them, see Hamernjk, *loc. cit.* Berard, "Mémoire sur un point d'anatomie et de physiologie du système veineux," etc., *Archives générales de médicine*, 1830, tome xxiii. p. 169. Ringer, S., and Sainsbury, H., "Pulsation and Murmurs in the Great Veins of the Neck," etc., *Lancet*, Nov. 28 and Dec. 5 1891.

[2] Reid, John, "On the Effects of Venesection in Renewing and Increasing the Heart's Action under Certain Circumstances," *Edin. Med. and Surg. Journ.* 1836, vol. xlv. p. 367.

[3] Riegel, F., *Deutsches Archiv f. klin. Med.* Bd. xxxi. p. 20.

[4] François-Franck, "Variation de la vitesse du sang dans les veines sous l'influence de la systole de l'oreillette droite," *Archives de Physiologie normale et pathologique*, 1890, tome ii. p. 317.

[5] Bamberger, "Lehrbuch der Krankheiten des Herzens," p. 97. Wien, 1887.

and permit the reflux of blood, and with subsidence of the turgidity again become quite competent. Apart from the question of competency of the jugular valves, there can be no doubt that stasis has a distinct modifying effect upon the character of the pulse tracing, but to what extent it is impossible to determine. By the graphic method here employed all movements are registered, and where the current may be continuous, but where variations in the rapidity produce variations in the distension and size of the veins, these variations in the size of the vein will be registered as distinct movements,—inasmuch as the method employed records only variations in the volume of the veins. But as such variations are caused by the same forces that produce an actual backward movement, no difficulty is experienced in recognising the cause of both. It need further only be said that no attempt is here made to determine what portion of the curves are due to actual backward currents and what are due to variations in the distension of the veins.

Although with one exception the tracings of the pulsations in the veins here given have been obtained from the internal jugular vein (usually the right), sometimes the movements have been observed in the subclavian veins (Case 6), in the small veins of the arm (Cases 3 and 42), in the veins of the face (Case 1), and in the superficial veins of the thorax (Cases 13 and 45). In these cases the movements were very distinct, but could be rendered more evident by the method employed by King of fixing a thread of sealing wax to the skin covering the vein, when the distal extremity of the thread executes greater excursions; or they may be rendered more visible by employing tiny specks of glass mirrors as used by Czermak and Hippesley.

Section VII.—The Forms of the Venous Pulse, and the Causes of their Production.

For the sake of convenience the pulsations in the veins may be divided into two classes, those in which the wave produced by the contraction of the auricle is a marked feature, and those in which the auricular contraction is no longer represented, only the wave produced by the ventricular contraction being present. The former will hereafter be spoken of as the "auricular venous pulse," and the latter as the "ventricular venous pulse." There is no sharp line dividing them, and while the auricular wave may disappear from the venous pulse the ventricular wave is practically always present, even in the more characteristic auricular venous pulsations. By the growth of this ventricular wave the one form becomes transformed into the other, and within certain limits the ventricular represents thereby a more advanced stage of engorgement than the auricular. Speaking generally, these two forms are those that have hitherto gone under the names of the normal, negative, or false venous pulse (auricular), and the true or pathological venous pulse (ventricular).

The changing of medical terms, is, at all times, to be deprecated, and when undertaken there should be some very cogent reasons for so doing. In the present case the names applied to the venous pulses carried with them ideas at total variance with the actual facts. The one is as true a pulse as the other; both are often equally abnormal. The names I give them predicate nothing as regards their relative importance, and merely refer to their most characteristic features and their causes. It would be better to do without any distinctive naming, were it not for the fact that the names I suggest are of great convenience, especially as regards the matter of brevity, in referring to certain salient points in the discussion of this subject.

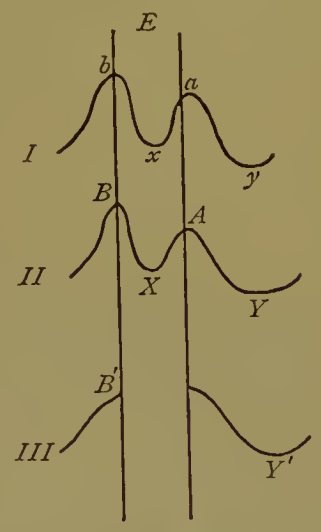

FIG. 13.—Diagrammatic representation of the venous pulse of the auricular type, and the forces producing it, during one cardiac revolution. (I) The venous pulse waves; (II) The pressure curve in the auricular cavity; (III) The pressure curve in the ventricular cavity while it is in communication with the auricle. The space *E* included within the perpendicular lines represents the time of ventricular systole (from the time of the closure to the opening of the tricuspid valves), during which period the ventricle ceases to participate directly in the changes in the auricle and veins (hence the ventricular curve is not represented during this period).

If it be borne in mind that the venous pulse may only make its appearance in those cases where there is abnormal distension of the right side of the heart and adjacent great veins, and that with this distension the dilated venæ cavæ do not contract as efficiently as in health, but permit at all times a freer communication between the auricle and the veins, the consideration of what the venous pulse is becomes much simplified. As a matter of fact, the veins and the auricle are practically one cavity. Hence variations in pressure occurring in one portion of the cavity will be reflected to all parts of the cavity. It follows, then, that a rise of pressure in the auricle is represented by a rise of pressure in the veins, which here manifests itself as a distinct, visible pulsation. A fall in the auricular pressure will likewise be reflected in the veins, giving rise to a negative wave. These variations in pressure arising in the auricle will be reflected along the veins till the factors opposed to their transmission overcome the force that produces them. In order to appreciate the time of the occurrence of the waves in the veins, and the factors producing them, it is only necessary to consider the variations of pressure in the auricle, and the causes producing the variations will be the causes producing the venous pulse. But, in addition, during the ventricular diastole, while the tricuspid valves are open, the ventricular cavity is likewise in free communication with the auricle and the veins. Variations of pressure will occur in the ventricular cavity during this period

at the same time, and be of the same nature, as the variations in the auricle and vein. If, then, these variations in the different cavities be represented diagrammatically, it will be found that while the cavities are in free communication the pressure rises and falls in all three cavities at the same time.

If we consider the auricular pressure curve (II, Fig. 13), there will be found to be a rise in pressure during the auricular systole *B*. At the same time also there is a rise in the ventricular pressure *B'*, and a wave in the venous pulse *b*. In the auricular pressure curve this is followed by a fall *X* during the auricular diastole. In the ventricular curve (III), there is at the beginning of the fall a sudden stop due to the closure of the tricuspid valve, the ventricle now ceasing to be in communication with the other two cavities. There is a fall *x* in the venous tracing, coincident with the auricular diastolic fall *X*. Towards the end of the ventricular systole, the auricular pressure again rises, and the rise is represented by a wave in the venous pulse *a*. The ventricular cavity is still shut off from the others. At the end of the ventricular systole the auricular pressure curve shows another fall *Y*, reflected in the venous pulse as a depression *y*, and, the tricuspid valve being now open, also present in the ventricular curve *Y'*. These variations in the venous pulse practically give rise to the curves taken from the jugular veins during a cardiac revolution, in the form which I have called the auricular venous pulse. What the forces are producing these variations in pressure, and how they are modified, will be the main subject of our inquiry.

FIG. 14.—Diagrammatic representation of the venous pulse of the ventricular type (I), and of the force producing it —the ventricular systole (II). The tricuspid valve being incompetent, the variations of pressure in the ventricle are reflected into the veins during the whole cardiac revolution. The space (*E*) enclosed within the perpendicular lines represents the time of the ventricular systole.

If we consider the condition in advanced regurgitation, the question will be found to be much simpler. As an active agent, impressing its individual movements on the neighbouring cavities, the auricle has ceased to be effective. It evidently reflects, passively, the variations in pressure produced by other agencies, and for the moment may be left out of account. In this condition of the venous pulse—the ventricular form—the tricuspid valves are greatly incompetent, and hence the ventricular cavity and the jugular vein are in free communication, and the variation of pressure in the ventricle is represented during the whole cardiac revolution in the vein. As there is, broadly speaking, but one great rise during ventricular systole, and a great fall during diastole, so

in the venous pulse there is but one rise and one fall during the cardiac revolution (Fig. 14).

(A certain analogy might be drawn between the venous pulse with incompetent tricuspid and the arterial pulse where the aortic valves are widely incompetent; in both cases there is a great rise during ventricular systole, and a great fall during the ventricular diastole.)

Section VIII.—The Individual Elements of the Venous Pulse.

1. *The Auricular Wave* (*b* in all the tracings).[1]—When tracings of the venous pulse are taken synchronously with the arterial pulse or apex

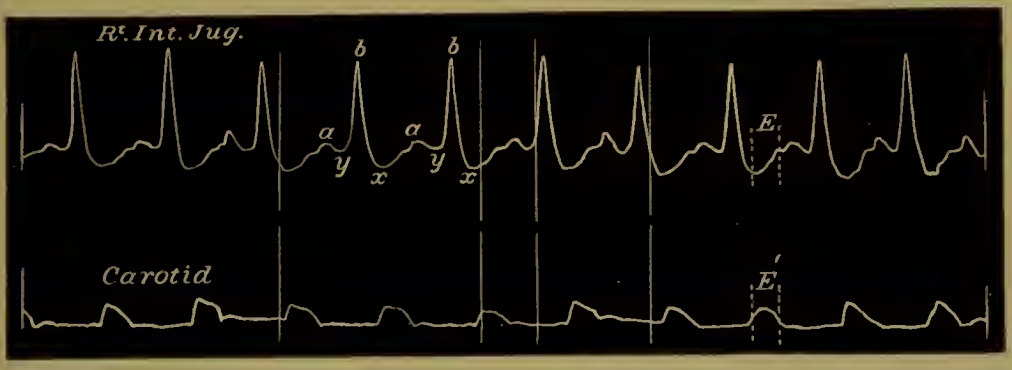

Fig. 15.—Tracing of venous pulse taken synchronously with the carotid pulse; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression; *y*, ventricular depression; *E* and *E'*, duration of the outflow through the arterial orifices of the right and left ventricles (Case 2).

beat, this wave is readily recognised, preceding as it does the effects of the ventricular systole (Fig. 15).

In the great majority of patients in whom the venous pulse is well marked, this wave is a very characteristic feature. It can very frequently be recognised on inspection, comparison being made at the same time by palpation of the carotid pulse. Its occurrence at a period distinctly prior to that of the carotid can often be readily demonstrated. Considering the frequency of the pulsation, and its very distinctive character, it is wonderful that so many experienced observers have either not recognised it or have denied the possibility of its existence. The reason for this has undoubtedly been the view that the contraction of the auricle represents such a feeble force as to be incapable of driving the blood backwards. Yet it is very important to recognise the force with which the auricle contracts, inasmuch as there is a distinct relation between its power of contraction and its capacity for dilatation, which

[1] In all the tracings the letters employed always bear the same meaning, viz., *b*, auricular wave; *a*, ventricular wave (*a'*, ventricular wave before the closure of the pulmonary valves); *c*, arterial wave; *x*, auricular depression; *y*, ventricular depression; *E*, time occupied by ventricular outflow though arterial orifices in vein or liver pulse; *E'*, time occupied by ventricular outflow through arterial orifices in apex beat or sphygmogram.

relation lies at the root of the proper understanding of the changes that take place in advanced heart disease. Experimental observations have demonstrated the force with which it can contract, and, clinically, we have evidence of the power of the left auricle in distending the left ventricle and making its presence felt in the apex beat, as in Fig. 137. Hilbert[1] gives a tracing of an apex beat with a very large auricular wave.[2] In cases of ossification of the ventricle, Burns[3] considers that the auricle had sufficient power to drive the blood into the arteries, and this idea receives some support from an observation made by Williams,[4] who during an experiment observed that the auricle continued to beat while the ventricle stood still and the circulation was maintained, and arteries bled on being cut, the blood, however, flowing in a steady stream. That the auricular contraction can affect the blood in the aorta appears demonstrated from some of Hürthle's tracings.[5] When the blood pressure was low, there was a distinct rise in pressure in the aorta, produced by the auricular contraction. Although these observations refer mainly to the action of the left auricle, it is but reasonable to expect that the right possesses similar attributes. In all cases of marked venous pulse, where there is no organic disease or abnormal dilatation of the valvular orifices, the auricular wave is a characteristic feature, and its presence is maintained so long as the venous pulse is manifested. It is important to note this, because cases will have to be considered in which the auricle ceases to manifest its activity in the venous pulse. That it can also make its presence felt in the peripheral veins is evidenced by such cases as the following:—

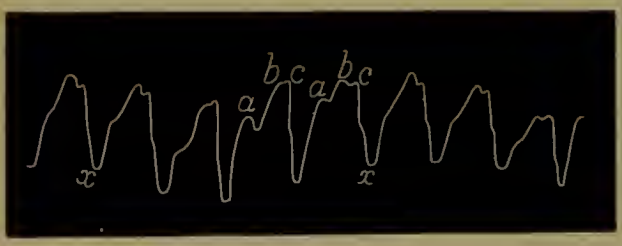

FIG. 16.—Phlebogram from the right internal jugular vein; *a*, ventricular wave; *b*, auricular wave; *c*, arterial wave; *x*, auricular depression (Case 1).

CASE 1.—Female, æt. 52; for many years given to indulgence in alcohol in secret, has been weakly for many months, the chief symptoms being great anorexia and swelling of the legs. There was slight enlargement of the heart's dulness to the right, and a loud systolic murmur at the base of the heart and over the middle of the sternum. There was marked pulsation of the auricular form in the internal jugular veins (Fig. 16).

[1] Hilbert, P., "Ueber die Ursachen des normalen und des krankhaft verstärkten Herzspitzenstosses," *Zeitschr. f. klin. Med.* Bd. xxii. Heft 1 and 2, fig. 14.

[2] On further consideration of the cause of the elevation in some cardiograms, as Fig. 137, said to be produced by the auricular systole, it does not appear to me quite established that the auricular systole is the whole cause. According to several observers it would appear that there is an active, if not even a powerful diastolic expansion of the ventricle, in which case this factor would produce a movement of the left ventricle nearly synchronous with, and of a character similar to, that said to be produced by the auricular systole in such cardiograms as those of Hilbert.

[3] *Loc. cit.*

[4] Williams, C. J. B., "The Pathology and Diagnosis of Diseases of the Chest," London, 1835, 3rd edition, p. 174, footnote.

[5] *Loc. cit.* fig. 230.

The timing of the events in this tracing was checked by the large recording apparatus. Towards the end of the patient's life pulsation was well marked in the veins of the face and scalp, and the day before her death the facial veins emptied during inspiration and filled and pulsated during expiration. The relative time of the venous pulse to the heart beat was maintained to the last. After death there was found great dilatation of the right side of the heart, with extensive fatty degeneration of the walls, but no valvular disease.

Even with cases of extensive valvular disease the auricular wave may persist even to the end. The following two cases of tricuspid stenosis show this wave very characteristically:—

Case 2.—Female, æt. 29; has been under my observation for 2 years. She suffers from shortness of breath on exertion, and latterly from severe attacks of pain striking from the front of the left chest down the inside of the left arm to the little finger, with great tenderness of the skin over the area of pain after the attack has subsided. She had erysipelas in the face 11 and 9 years ago, and since then she has been short of breath, and with a tendency to swelling of the legs. The face has the glazed, puffy appearance of the solid œdema following erysipelas. The pulse is small, quick, and regular. There is a large pulsating swelling on either side of the neck near the sternal end of the clavicle, but there is no distinct pulsation in the veins above these swellings. When compared with the carotid pulse there are found to be two distinct movements, the one larger than the other, to each carotid pulse, and the larger movement can be observed to precede that of the carotid pulse (Fig. 15).

The area of cardiac dulness is increased, extending transversely $1\frac{1}{2}$ in. to the right beyond the middle line. There has always been present a long murmur, presystolic in time, heard best at the apex; and one shorter and rougher of a different character, heard best over the middle of the sternum. This latter murmur was occasionally absent at first, but is now a constant phenomenon. There is also a murmur, systolic in time, heard at the apex. At the base the second sound is reduplicated. No murmur is to be heard in the carotids, but there is a distinct though dull sound heard over the pulsating swelling in the neck synchronous with the pulsation and preceding the carotid pulse. The liver dulness extends 2 in. below the level of the ribs; the liver can be felt pulsating distinctly (Figs. 32 and 33).

The diagnosis in this case is stenosis of the mitral and tricuspid valves. The two presystolic murmurs are so distinct, that when the heart is quiet, the opportunity for distinguishing them is readily afforded. In all likelihood there is also disease of the aortic valves. Although there is no murmur heard in the aortic area, the fact that all these valves are usually diseased when the tricuspid is affected (Case 3), and the fact of the appearance of angina pectoris—which from Nothnagel's[1] statistics, with which my own agree, is an extremely rare phenomenon in disease of the auriculo-ventricular valves, though frequent in disease of the aortic valves—leaves little doubt in my mind that all these orifices are affected. The auricular wave is such a prominent phenomenon in this case, both to the eye and in the tracings, that I have very little doubt that the auricle is here greatly hypertrophied.

[1] *Zeitschr. f. klin. Med.* 1891, vol. vii.

Case 3.—Male, æt. 29; examined September 1892. Complains of shortness of breath and swelling of the legs. He had rheumatic fever when he was 10 years of age, and has suffered much from shortness of breath and other symptoms of cardiac failure ever since. There is marked pulsation over the inner ends of the clavicles, particularly the left, also in both internal jugular veins, both axillary veins, the veins at the bend of the elbows, and in the back of the hands. The femoral veins also pulsate. The pulsation shows two movements for one arterial pulse, and the tracings show that one of these distinctly preceded the apex beat (Fig. 17). The transverse area of the heart's dulness extends $1\frac{1}{2}$ in. to the right side of the middle line and 4 in. to the left. In the mitral area a murmur, presystolic in time, is heard. The first sound is followed by a murmur propagated into the axilla. Over the whole sternum there is a loud murmur following the second sound. In the aortic area there is a short systolic murmur heard also over the vessels at the root of the neck. The liver is enlarged, extending 3 in. below the level of the ribs, pulsating at double the rate of the arterial pulse.

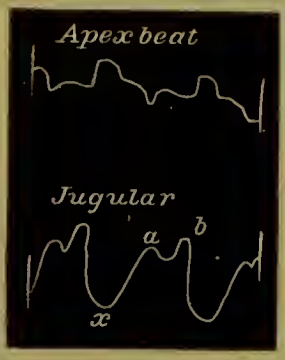

Fig. 17.—Tracings of apex beat and of the jugular pulse taken together; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression (Case 3).

The patient grew worse, the pulsation of the veins to a great extent disappeared, and he died with all the symptoms usually accompanying extreme heart failure. At the post-mortem examination the aortic, mitral, and tricuspid

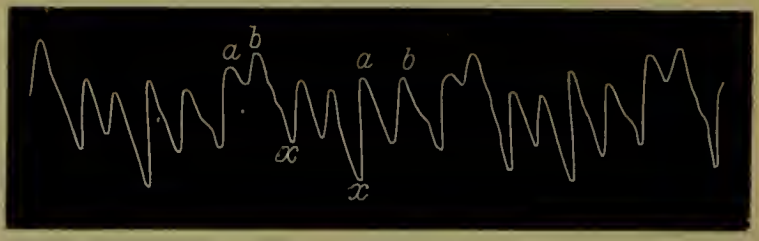

Fig. 18.—Tracing of venous pulse; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression (Case 3).

orifices were greatly narrowed, the valves around the two former being calcareous, and those around the tricuspid much thickened.

In connection with the marked jugular pulsation in these two cases, and their association with tricuspid stenosis, it is interesting to note that Gairdner[1] also observes the fact, though he does not distinguish the individual elements of the pulsation and their causes.[2]

In pregnant women, and during the debility following pregnancy, the auricular form of pulse is very common.

Case 4.—Female, æt. 26 years; seven months pregnant with second child. Is rather short of breath, otherwise is well. Except a slight systolic murmur

[1] Gairdner, W. T., "Clinical Medicine," Edinburgh, 1862, p. 602.

[2] Since the above was written, Professor Gairdner has published the results of the post-mortem examination of his case of tricuspid obstruction, in which there was found an auricular tumour blocking up the tricuspid orifice during the auricular systole. The surmise that the pulsation in the jugular, which was a prominent feature in the case, was due to the auricular systole is borne out by the consideration of Case 2, where the pulsations in the jugular veins has a remarkable resemblance to the description Gairdner gives of his case.—*Edinburgh Hospital Reports*, 1893, vol. i. p. 221.

at the base, there is nothing wrong with the heart. She was confined at full time, and made a good recovery. The venous pulse, which had been present for several months (Fig. 19), disappeared for a few days towards the end of the puerperium, but returned when she got up. Six months after confinement it was still present, presenting the same features, but not nearly so well marked.

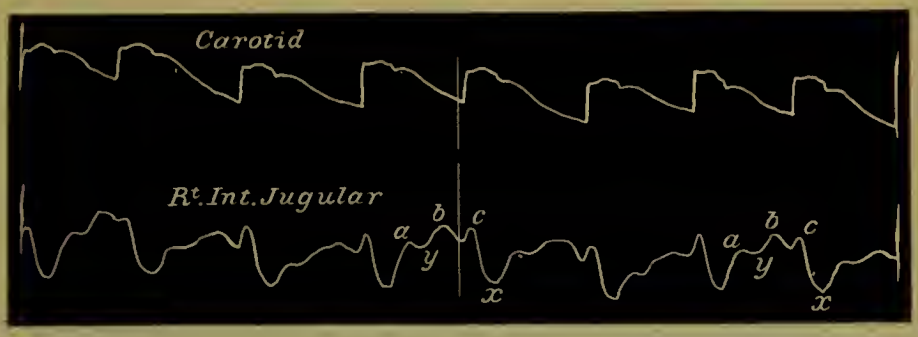

FIG. 19.—Tracings of carotid and jugular pulses taken at the same time; *a*, ventricular wave; *b*, auricular wave; *c*, arterial wave; *x*, auricular depression (Case 4).

The following two cases of mitral stenosis exhibit a very large auricular wave:—

CASE 5.—Female, æt. 12; examined 5th August 1892. Complains of weakness and shortness of breath. When 18 months old the patient suffered severely from bronchitis and whooping-cough, and has never been free from a cough since. It has been noticed that since she was 7 years old her ruddy face has become quite blue on exertion. Three years ago she was laid up with cough and shortness of breath.

Present state.—The patient is very thin, with dusky red countenance. The pulse is small, quick, and weak. The heart beats strongly against the chest wall with large diffuse apex beat in the fifth and sixth interspaces. The vertical dulness of the heart begins at the second interspace in the left parasternal line, and extends 1 in. to the right of the middle line, and to the left anterior axillary line. At the apex there is a loud murmur, replacing the first sound propagated into the axilla. Nearly the whole of the interval between the second and first sound is filled by a murmur running up to the first sound, and heard only in a limited area around the apex beat. There is also a high-pitched superficial systolic murmur over the middle of the sternum. There is no dropsy, no enlargement or pulsation of the liver. The jugular veins pulsate markedly, and give a tracing of the auricular type (Fig. 20). The patient's condition materially improved, and when last examined (24th August 1893), the pulsation in the jugulars was very small, and gave rise to but a very small tracing of the same type.

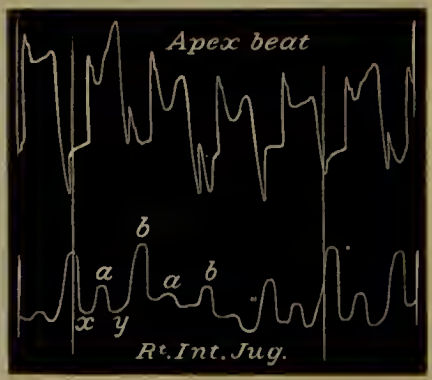

FIG. 20.—Tracings of apex beat, and of the right internal jugular pulse taken at the same time; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression; *y*, ventricular depression (Case 5).

CASE 6.—Female, married, æt. 32; examined 20th March 1892. The patient complains of shortness of breath and swelling of the legs and face. She has had two children, 11 and 7 years ago, and has always had good health

until last Christmas, when she began to be very short of breath, and her legs began to swell.

Present condition.—She lies in bed propped up, and suffers from great breathlessness if she lies flat. The legs are greatly swollen. There are some old scars in the neck, and brown stains from an old eruption on the back. The superficial veins crossing the right clavicles and the external jugular pulsate rapidly, and when timed with the radial pulse are found to beat twice for one radial pulse. A small vein over the eyebrows also pulsates. This double pulsation is also very well marked under both clavicles (Fig. 21), in the axilla, in the internal jugular, and in the veins in front of the right ear. The radial pulse is small and soft, 84 per minute.

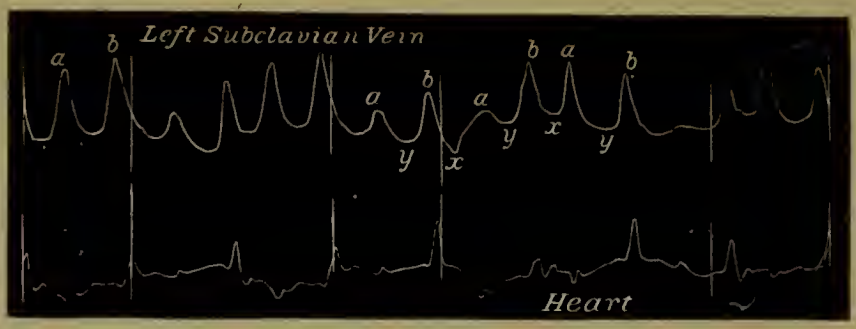

FIG. 21.—Tracings of pulsations in the left subclavian vein, taken at the same time as the heart impulse. The sharp elevations in the lower tracing indicate the beginning of the ventricular systole; *a*, the ventricular wave; *b*, the auricular wave; *x*, the auricular depression; *y*, the ventricular depression (Case 6).

There is a sharp shock communicated to the chest wall with each heart beat. No localised apex beat can be defined, and the sharp elevation in the heart tracing (Fig. 21) merely represents this sharp shock. The heart's pulsation is felt to the right of the sternum, and to the left as far as the posterior axillary line. The heart's dulness extends from 2 in. to the right of midsternum to 7 in. to the left. There is a murmur, systolic in time, heard over the whole area of cardiac dulness, but the character is somewhat different over the sternum from that heard outside the left nipple. The respirations are laboured. The base of the right lung behind is dull, and the breath sounds are indistinct, but a few crepitations can be heard. The left base is resonant, and there are crepitations with respiration. The abdomen is distended and resonant, with slight fluctuation, the walls being œdematous. The liver dulness does not extend below the ribs. The patient's condition steadily grew worse, and she died on 26th May 1892. At the post-mortem examination a long needle was passed into the left second interspace near the sternum. There was a large quantity of fluid in the pleural cavities. The heart was found greatly enlarged. The needle had penetrated the right auricular appendage. The right side of the heart was greatly dilated. The cone circumference of the tricuspid orifice was $4\frac{1}{2}$ in., that of the mitral $1\frac{1}{2}$ in. narrowed, and the segments of the valves adherent.

2. *The auricular depression* (*x* in the tracings).—With the diastole of the auricle there is a fall in the intra-auricular pressure, and, as a consequence, a great depression (a negative wave) in the venous pulse. The beginning of this period in the veins is shown to precede that of the carotid pulse. It is of particular importance to note that the fall begins before the carotid pulse, as in dealing with the same

phenomenon in the liver tracings it will be found to be the decisive point in one important matter of differential diagnosis. In many tracings the upper part of the fall is interrupted by the effect of the carotid pulse upon the air in the receiver. By this effect the auricular depression is often made easy of recognition—an abrupt interruption in the line of a great descent. The proximate cause of the auricular depression in the veins is of course the fall of pressure in the auricle. The fall is very considerable; according to some observers the pressure is below that of the atmosphere (Marey,[1] Porter[2]). The cause of this fall is, however, not easy to determine. The relaxation of the walls is rejected as having any effect. The suction action of the lungs upon the weak auricular walls and the retraction of the auriculo-ventricular valves during the ventricular systole have also been put forward as causes. Whatever the cause of the fall of pressure in the auricle may be, there is no doubt that it produces the auricular depression in the veins. In Marey's and in most of Porter's tracings of the intra-auricular pressure there is a slight interruption in the fall, but those of von Frey [3] do not show it. I have never found any interruption at this period in the tracings of the venous pulse, except that due to the carotid pulse. The important point to be considered in connection with the auricular depression is the manner in which its duration is limited. Generally speaking its size and duration bear a distinct relation to the vigour of the auricular contraction, and therefore to the prominence of the auricular wave in the venous pulse. This is to be expected, as the more powerfully the auricle contracts, and the more completely it evacuates its contents, the greater will be the period occupied by the auricular diastole. The converse likewise holds true: the feebler the contraction and the less complete the evacuation of the auricle, the shorter will be the period of auricular depression. The duration of the auricular depression is terminated by the appearance of the next wave. As the growth of this wave encroaches upon the auricular depression the cause of the limitation of the auricular depression will, therefore, best be considered in connection with the discussion of the rise and progress of the ventricular wave. I may here remark, however, that the effect of the auricular diastole in modifying the character of the wave transmitted from the ventricle has been entirely lost sight of by previous inquirers into this subject, and it is in the manner in which this modifying effect operates that the key for the solution of this problem of the nature of the venous pulse is to be found. Regarding the duration of the auricular depression it begins a little before, and lasts during a varying portion of the time occupied by, the ventricular systole, the variation depending to a certain extent on the degree of tricuspid regurgitation.

[1] Marey, E. J., "La Circulation du Sang," Paris, 1881, p. 114.

[2] Porter, W. Thomson, "Researches on the Filling of the Heart," *Journ. of Phys.* Oct. 1892, No 6, vol. xiii.

[3] von Frey, M., "Die Untersuchung des Pulses," Berlin, 1892, p. 88.

Before entering upon the consideration of the ventricular wave, it will be advisable to consider some points connected (1) with the incompetence of the tricuspid valves, and (2) with that period of time in the cardiac revolution between the closure of the semilunar valves and the opening of the auriculo-ventricular valves.

Section IX.—Incompetence of the Tricuspid Valves.

3. *The ventricular wave* (*a'* and *a* in all the tracings).—The natural tendency of the tricuspid valves to become incompetent has not received that recognition from clinical observers which the subject demands. That a slight form of tricuspid incompetence readily takes place was long ago demonstrated. John Hunter [1] and Richerand [2] looked upon the tricuspid valves as being naturally incompetent, while this incompetence was commented on by Adams,[3] and was established by the instructive series of experiments and investigations of Wilkinson King, already referred to. In his experiments on the dead heart King demonstrated that, with moderate distension, the tricuspid valves readily permitted the escape of water through the tricuspid orifices. Pursuing his inquiry into the condition prevalent in the lower animals he demonstrated that the safety-valve function of the tricuspid was necessary to the existence of those animals that had to suspend respiration for long intervals (divers, seals, etc.).

King's observations on the dead heart have been confirmed by the subsequent observations of Gairdner,[4] Luton,[5] Gibson,[6] and Krehl,[7] and the like readiness to incompetence has been demonstrated on the living animal by François-Franck [8] and Roy and Adami.[9]

Although, clinically speaking, the slighter forms of tricuspid incompetence are not generally recognised, yet several observers do distinguish certain characteristic phenomena. These are mainly the increase in the area of the heart's dulness, and the appearance of a tricuspid systolic murmur, whose situation of greatest intensity is found to vary in the description of each writer. But the effect of this

[1] *Loc. cit.* p. 179.

[2] Richerand, A., *loc. cit.* p. 167.

[3] Adams, Robert, *loc. cit.* p. 437.

[4] Gairdner, W. T., "On the Action of the Auriculo-Ventricular Valves of the Heart," *Dublin Hospital Gazette*, Oct. 1857, p. 295.

[5] Luton, A., article "Circulation" in the *Nouveau Dictionnaire de Médicine et Chirurgie pratiques*, tome vii. p. 175.

[6] Gibson, G. A., "Jugular Reflux and Tricuspid Regurgitation," *Edin. Med. Journ.* May 1880, vol. xxv.

[7] Krehl, L., "Die Mechanik der tricuspidal Klappe," *Archiv f. Physiologie* (Du Bois-Reymond's), 1889, p. 289.

[8] François-Franck, "Note sur la reproduction expérimentale des insuffiscances valvulaire du cœur," *Mémoires et Bulletins de la Société de Médicine de Bordeaux* (1882), 1883.

[9] Roy, C. S., and Adami, J. G., "Remarks on the Failure of the Heart from Overstrain," *Brit. Med. Journ.* Dec. 1888.

moderate incompetency on the venous pulse has practically never been recognised. Most writers hold with Riegel [1] that the only form of venous pulse due to tricuspid incompetence is that form I have called the ventricular. Other writers, as Bamberger, Allbutt,[2] Parrot,[3] Peter, and Gibson, describe the occurrence of the venous pulse in moderate tricuspid regurgitation, but they do not attempt to determine what particular form the venous pulse assumes. But it does show itself in a manner as striking as it is instructive.

Before entering into the discussion of the manner in which this takes place it will be necessary to consider a period of action in the heart's revolution, whose effects have not yet been appreciated. In Fig. 22 there is represented in a purely diagrammatic form a series of events occurring during a cardiac revolution.

The incidents represented here are—(1) the intra-auricular pressure curve (which, for the sake of simplicity, may also be considered as representing the venous pulse in view of the propositions already enunciated), (2) the apex beat curve, (3) the intra-ventricular pressure curve, and (4) the aortic pressure curve. The periods of time, *B* and *C*, embraced by the perpendicular lines, represent the time during which the aortic valves are open (*B*), and the auriculo-ventricular valves are shut (*C*). But it is to the period of time represented by the space *A*, included between the lines representing the closure of the semilunar valves, and that representing the opening of the auriculo-ventricular valves, that I would call attention. It is not pretended that this diagram represents the incidents faithfully. It is built up mainly from the study of somewhat similar diagrams given by Roy and Adami [4] and von Frey.[5] These authorities represent the ventricular pressure as remaining at a

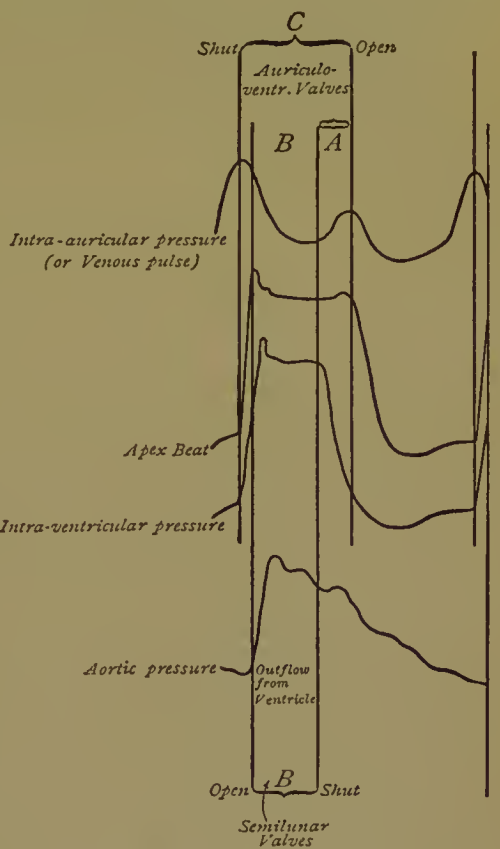

FIG. 22.—Diagramatic representation of a series of events, occurring during one cardiac revolution, to illustrate the intra-ventricular pressure during the period (*A*) between the shutting of the semilunar valves and the opening of the auriculo-ventricular valves.

[1] Riegel, F., "Zur Diagnose der Tricuspidal-insufficienz," *Berlin. klin. Woch.* 1886, No. 38, p. 621.

[2] Allbutt, T. C., "The Effects of Overwork and Strain in the Heart and Great Blood Vessels," *St. George's Hospital Reports*, 1870, vol. v. p. 23. [3] Parrot, *loc. cit.*

[4] Roy, C. S., and Adami, J. G., "Heart Beat and Pulse Wave," *The Practitioner*, 1890, p. 251, fig. 17. [5] von Frey, M., *loc. cit.* figs. 28 and 54.

considerable height after the closure of the aortic valves, although in von Frey's tracings the pressure has begun to lessen before this period. This period of relatively high pressure is found prolonged in most experimenters' curves, with the exception of Marey's,[1] Hürthle's,[2] and Porter's,[3] where the duration of this period is very much briefer than in the diagram. Hürthle expressly denies the existence of such a period of prolonged pressure after the closure of the aortic valves, unless exceptionally, as in poisoning by strychnine. But whether the duration of this period be long or short is not necessary to the argument. The essential fact is that at the moment after the closure of the semilunar valves, the pressure is relatively high in the ventricle. Even if it be admitted that the pressure in the ventricle is falling when the semilunar valves are closed, inasmuch as the closure of these valves is due to the arterial pressure rising higher than the ventricular, the closure must take place at a period when the ventricular pressure is high, and it is to this period, and the effect of the high ventricular pressure, that I particularly wish to draw attention. If, for instance, it be admitted for the moment that the tricuspid valves are incompetent, and the contracting ventricle is therefore driving blood in two directions, namely, into the pulmonary artery and through the tricuspid orifice, and a sudden cessation of the stream in one direction (towards the pulmonary artery) takes place, the flow is bound to be accelerated in the other direction, inasmuch as the force hitherto exerted in propelling blood in two directions is now entirely spent in the propulsion of blood in only one direction (through the tricuspid orifice). This result will be attained, however brief the period may be during which the ventricular pressure remains high, after the closure of the semilunar valves.

In the tracing of the auricular pressure there is a rise at about the time of the closure of the semilunar valves (Marey, von Frey, Porter). The period when this happens appears to vary. It is, in fact, at the termination of the auricular diastolic fall, and is in all probability due, as Porter suggests, to the flow of blood into the auricle from the veins. It is coincident in time with the wave I have called ventricular in the tracings of the venous pulse. The variations of the time of its appearance would depend upon the amount of blood that reaches the auricle during its diastole. In cases where there is but a moderate sized venous pulse, and the distension of the right heart but limited, it always appears at a time synchronous with that of the closure of the semilunar valves. If the flow of blood into the auricle be increased it will naturally appear earlier. During tricuspid incompetence the auricle will be filled more quickly, inasmuch as blood is pouring into it from two sources, namely, from the venæ cavæ and from the ventricle. It

[1] *Loc. cit.* fig. 50.

[2] Hürthle, K., "Beiträge zur Hämodynamik," *Pflüger's Archiv f. die gesammte Physiologie*, 1891, Bd. xlix. p. 29.

[3] *Loc. cit.*

follows, then, that the ventricular wave will appear earlier than usual. But, as I have already remarked, with closure of the pulmonary valves, the back flow from the ventricle is suddenly increased, there will then be reflected a sudden increase in the amount of blood in the auricle, which will also be reflected into the veins, and this is exactly what can be seen in many tracings.

In cases of moderate distension of the right heart there is usually but a small rise in the venous pulse synchronous with the closure of the pulmonary valves, as in Fig. 23. This may be considered as the

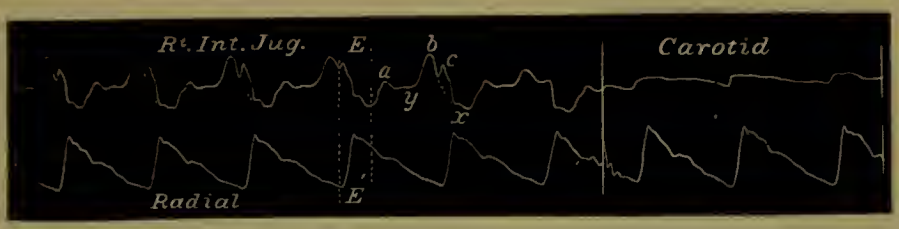

FIG. 23.—In the first part of the tracing the pulsation in the right internal jugular vein is taken synchronous with the radial pulse, in the latter part the carotid pulse is taken at the same time as the radial. *E* and *E'*, duration of ventricular outflow, right and left, through arterial orifices on venous and arterial pulses; *a*, ventricular wave; *b*, auricular wave; *c*, arterial wave; *x*, auricular depression. The dotted line between *b* and *x* indicates, probably, the true venous curve (Case 11).

earlier or rudimentary form of that ventricular wave which, in advanced tricuspid incompetence, ultimately becomes the most striking feature in the venous pulse. If tricuspid incompetence be moderate, *the effects of the back flow will be spent in the first instance in dilating the auricle and the great veins. It follows that a venous pulse cannot appear in the peripheral veins, such as the jugular, till there has been advanced distension of the more central veins; in other words, the regurgitant effects will be expended in distending the auricle and large venous trunks.* With greater distension the effects of the auricular and ventricular contractions will be reflected further back. If the veins are full at the beginning of auricular systole, and the great veins distended so that the sphincter-like action of the venæ cavæ at their mouths is ineffective, the auricular systole will propagate a wave back into the veins. If the systole is effective, the auricular diastole will likewise be effective, so that when the ventricle contracts before the regurgitant blood can appear in the veins, it will first have to distend the auricle and great veins, *and it will only be the effects of the latter part of the ventricular systole that will be evident.* Thus it is that it frequently happens that the part played by the high ventricular pressure after the pulmonary valves are closed is alone evident.

This effect of the increase of distension of the right side of the heart is well illustrated in the tracing (Fig. 24). In this patient (Case 15) the venous pulse had been a well-marked phenomenon. It had, on the day that this tracing was taken, completely disappeared.

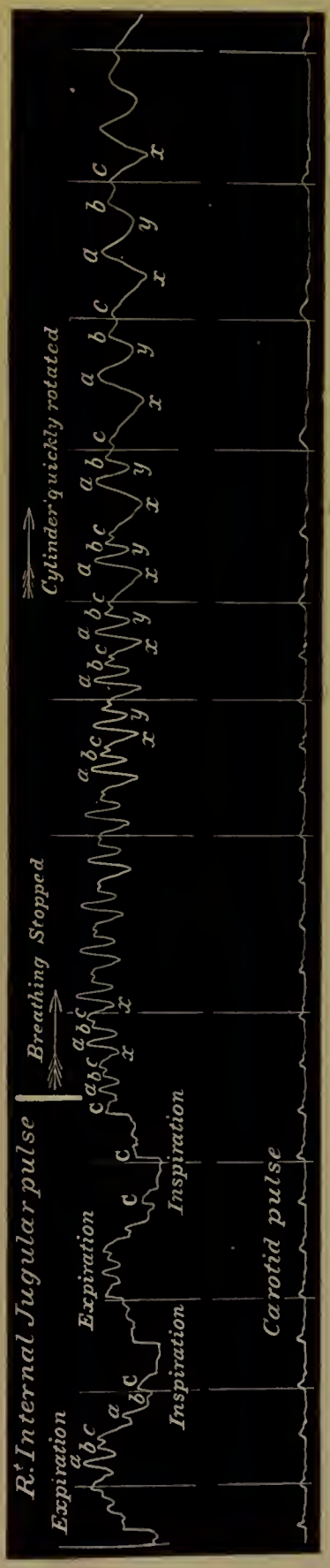

Fig 24.—Simultaneous tracings of the pulses in the right internal jugular vein and carotid artery. The venous pulse is at first only present during expiration. Two inspiratory and expiratory movements are recorded at the beginning of the tracing, and at the bottom of the inspiratory depressions there are only carotid pulse beats *C*, the venous pulse returning during expiration. When the breathing was stopped the venous pulse became more marked and continuous, being of the auricular type. Towards the end the cylinder was rapidly rotated in order to separate widely the events, and it is to be noted that the carotid pulse is always synchronous with the arterial waves *c*, and the carotid pulse beats *C C* at the bottom of the inspiratory depressions (Case 15). (Reduced one-half.)

By making the patient hold her breath, the venous pulse gradually returned. At first it was only evident on expiration. Thus, in the beginning of the tracing, two respiratory movements are recorded, the rise being due to expiration and the fall to inspiration. At the bottom of the two inspiratory falls there is evident in each one arterial wave (*C C*) without any evidence of the venous pulse. During expiration the venous pulse waves appear. When the patient held her breath the characteristic venous pulse occupied the remainder of the tracing.

The cause of the production of the venous pulse here is very readily understood. The main factors in producing the flow of blood through the lungs are the movements of respiration and the contraction of the right ventricle. If one of these be removed, more work will be thrown upon the other. Thus, then, when the patient held her breath the work of the right heart was increased. The tricuspid valve had long been incompetent, and the auricle and great veins greatly distended. In the progress towards recovery the distension had diminished. With increased work the ventricle now became distended, the tricuspid more incompetent, and the distension of the auricle and veins increased, so that the movements produced by the

auricle and ventricle became transmitted to a greater distance. Further, during inspiration the flow through the lungs is more favoured than during expiration, and the decrease of the intra-thoracic pressure during inspiration favours the greater distension of the intra-thoracic veins; hence it is that the venous pulse is not transmitted back so readily as during expiration. This increase in the tricuspid regurgitation during expiration has been experimentally demonstrated by François-Franck,[1] and during embarrassment of the respiration by Chauveau and Faivre as already quoted.

Houston[2] showed very clearly the natural provision for this venous stagnation in the case of diving animals, and Reid's[3] experiments on animals suffocated demonstrated the backward flow of blood due to the contraction of the heart. I have also observed blood being pumped back through the cut external jugular vein in an operation in the neck, where the patient under chloroform had ceased to breathe while yet the heart contracted. There is one point connected with tricuspid incompetence which is far from clear. If distension of the right ventricle produces incompetence of the tricuspid, does that incompetence last during the whole systole or only during the earlier part of the systole, until the abnormal distension is overcome? I can find no clear answer to that question. From Richerand's and Krehl's statements I should infer that the blood escapes naturally during the whole period of ventricular systole. In one of Gibson's experiments the whole of the water escaped backwards from the ventricle, on the maintenance of the intra-ventricular pressure. But this is far from being decisive, inasmuch as the papillary muscles by their contraction accommodate the position of the valves to the size of the contracting ventricle; this effect, of course, being lost in the dead heart. This point is important in its bearing on the explanation of the effects of closure of the pulmonary valves. If the tricuspid valves are competent at the time of the closure of the pulmonary valves, then no regurgitation could take place. It must be remembered, however, that the right ventricle never completely empties itself of its contents (Hope, Williams, Landois, Chauveau and Faivre, Roy and Adami[4]). With the engorgement of the right heart, the residual blood after the closure of the pulmonary valves will be increased in amount. The function of the papillary muscles is to close the tricuspid orifice. These muscles relax while yet the intra-ventricular pressure is high (Roy and Adami[5]). While the relaxation may not be accompanied by incompetence under normal circumstances, yet with marked distension it seems very probable that regurgitation will take place. Although I cannot

[1] François-Franck, "Recherches expérimentales sur le spasm bronchique et vaso-pulmonaire dans les irritations cardio-aortique," *Archives de Physiol. norm. et path.* 1890, tome ii. p. 547.

[2] Houston, J., "On the Peculiarities of the Circulating Organs in Diving Animals," *British Association Reports* (*Dublin*), *Medical Section*, 1835, p. 81.

[3] *Loc. cit.* [4] *Brit. Med. Journ.* Dec. 15, 1888.

[5] "Heart Beat and Pulse Wave," *The Practitioner*, 1890, p. 93.

appeal to any definite facts to explain the condition at this period, yet we have evidence of a very powerful force operating exactly at the period of time when the pulmonary valves close; and if the factors that could be operative during this period (see space *A* in the diagram, Fig. 22) be considered, the only one capable of producing this effect is the one I have described, namely, the high intra-ventricular pressure at the time of closure of the pulmonary valves, transmitting a wave back through the distended auricle and great veins into the jugulars. The following case illustrates this point:—

Case 7.—Male, æt. 32; examined October 31, 1892. He has been in bad health for two years, complaining chiefly of shortness of breath on exertion and great weakness. He is pale, and the body is moderately well nourished. The pulse is small, quick, and soft. Temperature, 100°. Marked double pulsation of both internal jugular veins. Heart dulness slightly increased to the right. A loud systolic murmur is heard over the whole heart, loudest over the middle of the sternum. The liver is enlarged, and extends 3 inches below the ribs. The patient died on 2nd November; no post-mortem examination was permitted.

The tricuspid incompetence was a marked feature in this case, and although its direct evidence is dependent only on the results of the physical examination, yet my examination receives the valuable confirmation of the opinion of Dr. Leech of Manchester. I saw the patient only a few days before his death, he having been recommended to me by Dr. Leech, who in his letter to me discussed the patient's condition, and remarked on the tricuspid incompetence, and concluded with the view that the diagnosis lay between cancer of the liver and pernicious anæmia (on account of certain changes in the blood). The tracing taken by the phlebograph (Fig. 25) represents a strikingly high ventricular wave *a*. The timing of the events is correct, having been verified by tracings with Knoll's polygraph, but this tracing is given in preference, as it was taken under more favourable circumstances. It was not taken simultaneously with the radial, but on the same visit, and the paper travelled at the same rate as when the radial pulse was inscribed. I have given them together that a comparison may be made of the occurrence of the various events. The high ventricular wave *a* occurs just at the time of the closure of the pulmonary valves. The auricle still contracts, giving rise to a marked wave *b*, and in consequence to a marked auricular depression *x*.

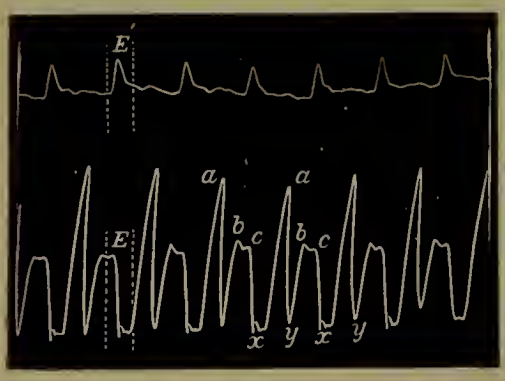

Fig. 25.—Tracings of the radial pulse, and of the pulse in the right internal jugular vein, to show the high ventricular wave *a* after the cessation of the ventricular outflow through the arterial orifice *E* (Case 7).

Very frequently the ventricular wave makes its appearance earlier than the time of the closure of the semilunar valves, and the time of its appearance serves in a measure to indicate the extent of the tricuspid incompetence. In the auricular form of venous pulse, the first part of the ventricular wave is never fully shown, and the latter part of the wave before the closure of the pulmonary valves generally appears as a gradual rise. The time of the pulmonary valve closure is indicated usually by a sudden increase in the size of the ventricular wave (Fig. 26).

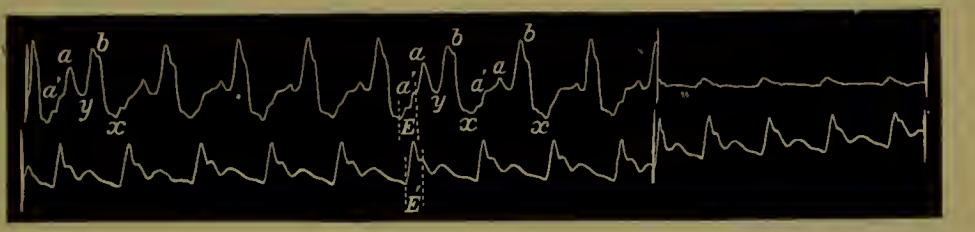

FIG. 26.—Pulsation of the left jugular bulb in the first part, and of the carotid in the second part of the upper tracing, taken at the same time as the radial pulse (lower tracing). The sudden rise after the cessation of the ventricular outflow through the pulmonary orifice *E* is shown : *a'* represents the ventricular wave before, and *a* after, the closure of the pulmonary valves ; *b*, the auricular wave ; *x*, the auricular depression (Case 2).

When the auricular wave has disappeared, and also, consequently, the auricular depression, the auricle, no longer dilating, offers no impediment to the backward flow from the contracting ventricle,—in such cases the tricuspid valves are greatly incompetent,—and the pulse then appears in the jugulars, at the same time as the carotid pulse, and, synchronously with the closure of the aortic valves there is a secondary wave due to the closure of the pulmonary valves.

This increase in the size of the ventricular wave, diminution in the time occupied by the auricular depression, and disappearance of the auricular wave, may be diagrammatically represented as in Fig. 27. These curves represent the venous pulse in six different cases, according to the degree of tricuspid incompetence.

These are practically the forms the venous pulse assumes in Cases 11, 29, 12, 16, and 21. The space *E*, included within the perpendicular lines, represents the duration of the outflow through the pulmonary orifice during the ventricular systole. The portion of the tracing after the latter of the two lines represents the wave caused by the increased flow, due to the high pressure in the ventricle after closure of the semilunar valves. The time occupied by the auricular diastole is gradually obliterated, while, on the other hand, the time occupied by the ventricular depression increases.

Before entering more fully into the consideration of the manner in which these changes occur, it may be well to consider the last phase of the venous pulse, the ventricular depression, and the liver pulse. The

liver pulse offers a means of analysing the events that occur in regurgitation from the heart, in a manner different from the pulse in the jugular veins, and the one pulse supplements the other in a very striking and instructive manner.

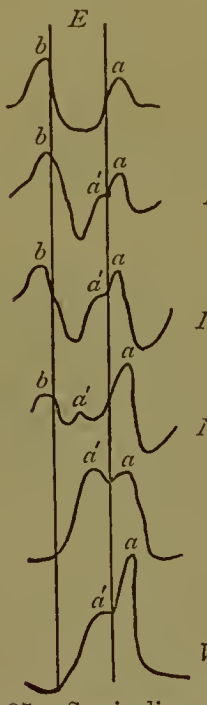

FIG. 27.—Semi-diagrammatic representation of the transition of the venous pulse from the auricular type into the ventricular. The space *E* represents the duration of the ventricular outflow through the pulmonary orifice, and the lines enclosing it therefore represent the time of opening and of shutting of the pulmonary valves. Coincident with the growth of the ventricular wave *a'* and *a*, there is a diminution and ultimate disappearance of the auricular wave *b*. These figures (with the omission of the arterial wave) are obtained: I. from Fig. 23 (Case 11), II. from Fig. 90 (Case 29), III. from Fig. 46 (Case 12), IV. from Fig. 55 (Case 16), V. from Fig. 74 (Case 21), VI. from Fig. 72 (Case 21).

4. *The ventricular depression* (*y* in all the tracings).—The cause of the fall in the venous pulse after the ventricular wave, appears very easy of explanation. After the ventricular systole there is a great and rapid fall in the intraventricular pressure, usually below that of the atmosphere. We have seen that at this time the pressure in the auricle has begun to rise. As soon as the intraventricular pressure falls below that of the auricle, as a matter of necessity the contents of the auricle must flow into the ventricle, and according to Goltz and Gaule[1] and De Jaeger[2] a negative pressure appears in the auricle as well as in the ventricle. A fall in the pressure in the auricle must ensue, a depression in the jugular pulse follows, and thus is produced the ventricular depression (see Figs. 13 and 22). Certain changes may take place in its size. It may disappear as in the auricular form of venous pulse, during rapid action of the heart. (In the meantime the consideration of the effects of rapid heart action will be deferred.) It may increase in size. As the ventricular wave grows at the expense of the auricular depression, so, in a sense, does the ventricular depression grow at the expense of the auricular wave. The greater the effect the systole of the auricle or the ventricle has upon the venous pulse, the more marked will be the evidence of the diastole of the one or of the other. As the auricle becomes less capable of independent action, the period occupied by the ventricular diastole increases, until finally, when the auricle has ceased to manifest its influence in the vein, the ventricular diastole fills the whole period between the end of one systole

[1] Goltz, Fr., and Gaule, I., "Ueber die Druckverhältnisse im Innern des Herzens," *Archiv f. die gesammte Physiologie* (*Pflüger's*), 1878, Bd. xvii. p. 100.

[2] De Jaeger, S., "Ueber die Saugkraft des Herzens," *Archiv f. die gesammte Physiologie* (*Pflüger's*), 1882, Bd. xxx. p. 508.

and the beginning of the other. When the venous pulse reaches this stage there is only manifested the ventricular systolic wave and ventricular diastolic depression, in fact the characteristic ventricular venous pulse.

The explanation thus given of the cause of the ventricular wave and depression in the ventricular form of the venous pulse is practically that accepted by all writers, with the exception that the division of the former into two periods is not recognised. In many of the waves in the ventricular form there is a saddle-shaped depression on the summit, as in Fig. 70. The cause of this has been disputed. Bamberger,[1] who first described it, considers that the second rise is due to the contraction of the papillary muscles, accelerating the flow, and in some cases as due to the recoil of the instrument. Geigel and Friedreich[2] considered that it was entirely artificial, the depression being caused by the recoil of the instrument (Marey's sphygmograph). Riegel[3] timed the tracing carefully, and found that the second rise followed immediately on the closure of the aortic valves, and attributes it to the closure of the pulmonary valves. He does not say how the pulmonary valves would produce such a wave. My results agree with his in the time of the events; the method of the production will be illustrated by a series of cases.

But it is as to the cause of the production of the ventricular wave in the auricular form of the venous pulse, that a great divergence of opinion prevails. Potain[4] finds it in his tracings, but gives no explanation of its presence, merely remarking that the fall I have called the ventricular depression is due to the ventricular diastole. It is also present in Gottwalt's tracings, but he gives no definite interpretation. Riegel's[5] explanation is rather far fetched. He thinks that the widening of the aorta compresses the superior vena cava, and thus produces the ventricular wave. With the recoil of the aorta after the ventricular outflow has ceased, the aorta recedes from the vena cava, and the vena cava expands and draws in blood from the jugular veins. The fact that the ventricular wave and depression are marked features in many cases of liver pulsation sufficiently disposes of this interpretation. François-Franck,[6] who, more than any one, has endeavoured to demonstrate this subject by experimental investigations, is scarcely intelligible at this point. He considers that the ventricular wave is only a superadded element of variable importance. It is, according to him, but a temporary rise, coincident with the passing of the ventricle from a state of systole to one of diastole, and agrees with the time of a rise in auricular pressure on Marey and Chauveau's tracings. It is quickly replaced by a new

[1] Bamberger H., *Würzburger med. Zeitschrift*, 1863, Bd. iv. p. 232.

[2] Friedreich, N., *Deutsches Archiv f. klin. Med.* 1865, Bd. i. p. 268.

[3] Riegel, F., *Deutsches Archiv f. klin. Med.* 1882, Bd. xxx. fig. 18 *a*.

[4] Potain, *loc. cit.*

[5] Riegel, *loc. cit.*

[6] François-Franck, *Gazette Hebdomadaire de Méd. et de Chirurgie*, 1882, No. 14, p. 257; *Compt. rend. des Séances et Mémoires de la Société de Biologie*, 1882, p. 62.

depression (the ventricular of my tracings), due to the venous current being accelerated by the relaxation of the ventricles, such relaxation not being due to the qualities inherent in the ventricle itself, but due to the traction exercised on the walls of the ventricle by the elasticity of the lungs. And yet he gives tracings showing the ventricular wave and ventricular depression taken from the dog when the chest was opened and the elastic force of the lung abolished.[1]

Section X.—Pulsations of the Liver.

The pulsations of the liver can be divided into two groups, with attributes similar to those characteristic of the two groups of the venous pulsations, namely, an auricular and a ventricular. When a liver pulse and a pulse in the internal jugulars are present in the same individual they are of an identical form,—a pulse of the auricular form in the veins being always associated with a pulse of the auricular form in the liver, a pulse in the veins of the ventricular form being always associated with a pulse of the ventricular form in the liver; an auricular venous pulse never being associated with a ventricular liver pulse, or *vice versâ*. Generally speaking, the character, the form, and the time of the associated liver and venous pulses are the same. There are, however, a few minor differences, and these differences, being appreciated, give useful information in regard to the production of the various events, and they will be commented upon in the further development of the argument.

1. *Auricular form of the liver pulse.*—It has already been remarked in dealing with the arterial wave that the events occur practically at one and the same time in the pulse of the jugular veins and of the liver, and that from the liver tracings there is an absence of all indications of the arterial wave (Figs. 10 and 11). When such a liver pulse is taken synchronously with the carotid pulse, the beginning of the latter falls immediately after the auricular wave has reached its summit, and after the descending line of the auricular depression has begun

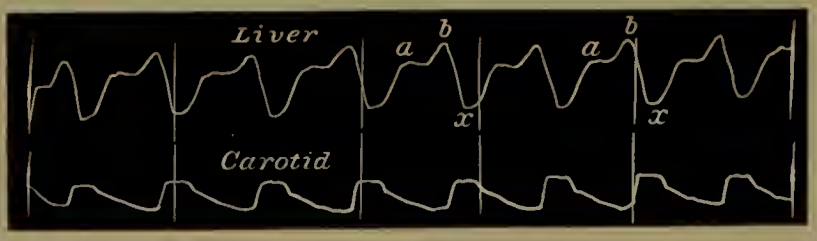

Fig. 28.—Tracings of the liver pulse, taken at the same time as the carotid pulse; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression (Case 46).

(Fig. 28). Then follows a characteristic depression during the ventricular systole, due to the diastole of the auricle *x*, with a rise towards

[1] *Gazette Hebdomadaire*, No. 14, *loc. cit.* fig. 4, p. 225.

the end of the ventricular systole *a*. The depression due to the ventricular diastole *y* is not always well marked, but still it is evident in Fig. 29.

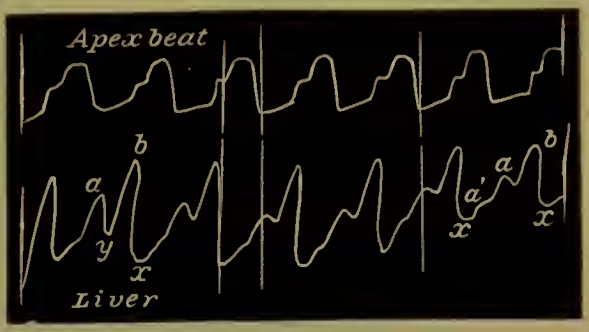

FIG. 29.—Tracings of apex beat and liver pulse taken synchronously; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression; *y*, ventricular depression (Case 41).

CASE 8.—Female, æt. 34, married. She was in good health till the third month of her first and only pregnancy, six years ago, when she became very short of breath. She was prematurely confined at the end of the sixth month, and during my attendance on her at that time I found out the condition of her heart. I have attended her since frequently. She has always been very weak and short of breath; her face often becoming quite blue on the slightest exertion. Two years ago I attended her, and noted then the pulsation in the jugular veins and in the liver. In May 1891 she had a smart attack of influenza, from which she made a good recovery.

Examined on 15th December 1892.—The pulse is small, weak, and regular. The jugular veins are full, and give a characteristic auricular tracing (Fig. 10). The whole præcordia heaves. The apex beat is in the fifth interspace outside the nipple line, and a purring tremor running up to the beat can be felt here. The area of the heart's dulness extends from 1½ inches to the right of the sternum to 2 inches beyond the left nipple. At the apex a rough murmur is heard running up to the first sound, which is followed by a soft, blowing murmur, heard in the axilla. Over the fourth and fifth costal cartilages a loud, roaring systolic murmur is heard, different in character from that of the apex. (Frequently this murmur has been absent when the heart is first auscultated, while the patient is in bed, but on exertion it is immediately developed.). At the base the second sound is markedly reduplicated and accentuated. The liver dulness extends 3 inches below the ribs, and gives a characteristic auricular liver pulse tracing (Fig. 30).

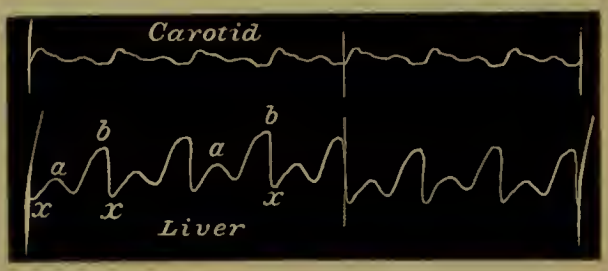

FIG. 30.—Tracings of carotid and liver pulses taken synchronously; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression (Case 8).

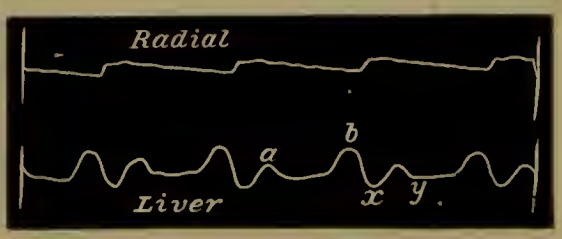

FIG. 31.—Tracings of radial and liver pulses taken together; *a*, ventricular wave; *b* auricular wave; *x*, auricular depression; *y*, ventricular depression (Case 8).

Examined again on 25th February 1893.—Practically no change, except that there is no pulsation in the jugular veins, while the liver is as it was at the last examination (Fig. 31).

I presume that it is because of the resistance offered by the liver to the distending force of the auricle and ventricle that the tracing is more rounded than that of the venous pulse. Fig. 32 is a tracing from the

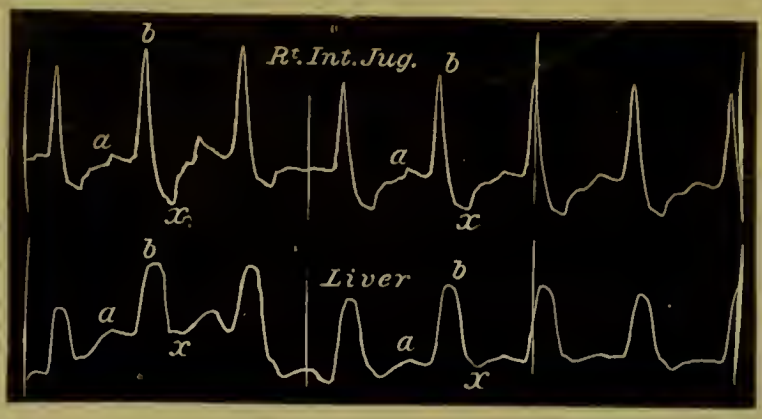

Fig. 32.—Simultaneous tracings of jugular and liver pulses ; *a*, ventricular wave ; *b*, auricular wave ; *x*, auricular depression (Case 2).

liver pulse of the patient with tricuspid stenosis (Case 2), and shows a distinct rounding of the curves. Still this partly depends upon the position in which one holds the liver receiver, for it is not always possible to get an identical tracing, although the main features are always recog-

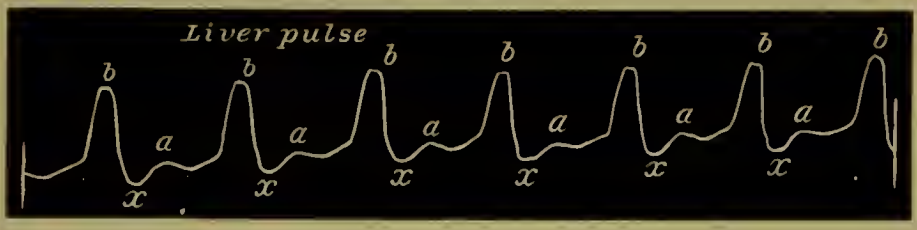

Fig. 33.—Tracing of liver pulse ; *a*, ventricular wave ; *b*, auricular wave ; *x*, auricular depression (Case 2).

nisable. Fig. 33 was taken from the same patient, and the resemblance to the venous pulse tracings in Figs. 15 and 32 is very obvious.

2. *Ventricular form of the liver pulse.*—In this form of the liver pulse the characteristic feature is the effect produced by the latter portion of the ventricular systole. Frequently there is but a slight rise due to the earlier portion of the ventricular systole, and the summit of the wave is reached considerably later than the beginning of the ventricular systole. In many cases, as in Fig. 34, no safe indication is given of the time of the pulmonary valve closure, even when this is well marked on the venous pulse, as in Fig. 131, taken from the same patient. Some-

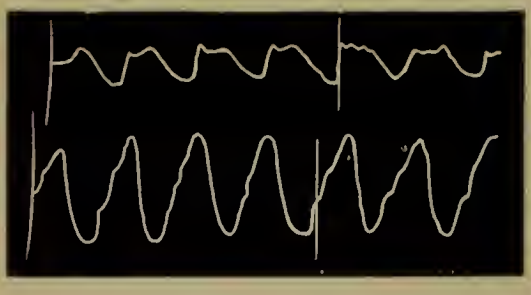

Fig. 34.—Tracing of carotid and liver pulses. The liver pulse is mainly ventricular systolic in time (Case 45).

times in some of the waves a small rise due to the earlier portion of the systole is detectable (a', Fig. 35).

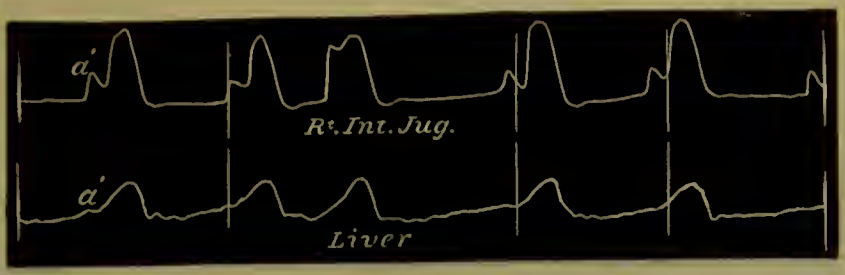

Fig. 35.—Simultaneous tracings of jugular and liver pulses, showing occasional faint indications of the first portions of the ventricular wave a' (Case 44.)

By this process of analysis readily provided for us it will frequently be possible to detect the effects of the ventricular systole after the outflow through the pulmonary orifices has ceased. When the latter portion of the ventricular systole is well marked in the venous pulse, it shows itself characteristically in the liver pulse, as in Fig. 36, where also there is a small wave due to the earlier portion of the ventricular systole.

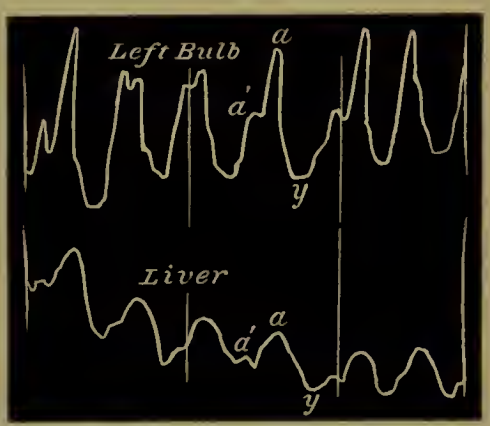

Fig. 36.—Simultaneous tracings of the pulsation in the left jugular bulb, and in the liver, showing the small size of the wave in the liver pulse due to the ventricular systole before the closure of the pulmonary valves a', and the relatively larger wave occurring after the closure of the pulmonary valves a; y, the ventricular depression, (Case 21).

In some of the liver pulse tracings the wave begins to appear earlier than the outflow from the ventricles. In such cases Riegel considers the first part due to the contraction of the auricle, and hence calls the liver pulse presystolic-systolic in time. I am disposed to doubt this explanation. On March 22, 1892, Fig. 34 was taken from the patient when the beginning of the liver pulse was practically synchronous with the beginning of the carotid pulse. (The lengthening of the systolic rise of the carotid pulse is not indicative of the time occupied by the ventricular outflow, for reasons explained in Section III.)

At this time the venous and liver pulses were evidently systolic in time. The patient's condition gradually became worse, the liver dulness extended below the level of the iliac crest; the tracing (Fig. 37) was taken with the receiver immediately above the pubes. The venous pulse had gone, the arterial pulse was too small to be suitable for a standard of time, and the position and character of the apex beat had changed. In fact there was distinct evidence of more advanced failure of the heart. Yet the liver pulse appears earlier. It would seem more reasonable to consider that the fall in the pulse of the liver is

due to the ventricular diastole; at the moment the diastolic suction ceases the liver begins to swell from mere stasis, such swelling be-

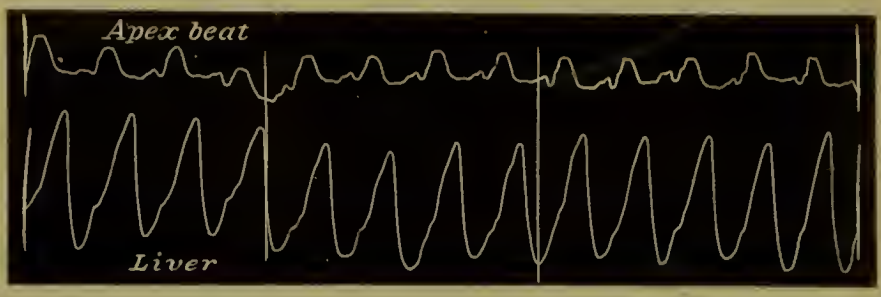

Fig. 37.—Tracings of apex beat and liver pulse, showing the duration of the liver pulse to be mainly systolic in time (Case 45).

ginning before the ventricular systole, and increasing by the regurgitation during the ventricular systole. This explanation seems more suitable than to assume a revival of the auricular action, long after the auricle has ceased to make its action manifest, while the condition producing the auricular paralysis had become exaggerated.

Section XI.—Mechanical Effects of the Heart Movement upon the Liver.

In reading records of clinical observations relative to the time of the various movements of the heart, veins, and liver, there is nothing that has surprised me more than the liability to error to which one is exposed when a preconceived idea as to what should happen is present in one's mind when the evidence of the senses is appealed to. Although liver pulsation is far from an uncommon occurrence, and although the time of its occurrence is regular and rhythmical, yet practically no clinical observations have been able to anticipate the results of exploration by the graphic method.

The idea has been present that certain actions of the heart, such as the systole of the right ventricle, would produce an impact upon the liver, so that a liver pulsation would be simulated. And numerous observations to that effect have been made. And yet if one considers what happens during the ventricular systole, it will be realised at once how impossible it is that such a thing should happen. Because the left ventricle, on account of its peculiar construction, raises its apex and impinges against the chest wall, it is tacitly assumed that all the other cardiac chambers do the same. For instance, we have abundant accounts in text-books of the pulsations of auricles and ventricles being recognised, and most accurate diagnosis based upon such hypothetical evidences; so in regard to the effects of the ventricular systole upon the liver—it is supposed to give rise to a spurious form of pulsation.

It will be necessary to glance for a moment at the movements of the heart, so far as they might affect the liver. I shall have occasion to

revert to the subject, so I will here merely touch upon the movements of the ventricles. Stokes and Hart found that the ventricles contracted in every direction, and the apex also retired from the finger.[1] In the report of the Dublin committee[2] the apex is described as being tilted up only—no movement occurring in any other direction, save shrinking during the ventricular systole. Von Ziemssen,[3] in the exposed heart of Frau Serafin, found that the apex moved to the right but not downwards. Skoda,[4] in an observation on the ectopic heart of an infant, describes a downward movement, but when the relative size of the hearts in Ziemssen's and Skoda's cases are considered, it will be seen that the opportunities of the former afforded the surer means of arriving at a safe conclusion. The movements of the apex beat to the right I have been able to demonstrate in cases of enlarged left ventricle, where, by pushing the button of a Knoll's polygraph into the upper part of the epigastrium, and to the left a very good tracing identical with that of the apex was obtained. Finally, Waller[5] describes the process succinctly as follows:—"This systolic shrinkage (of the heart during ventricular systole) is accompanied by a certain amount of twisting in consequence of the spiral disposition of the muscular layers which form the ventricles; the rotation is such that the heart twists round its long axis from left to right with the systole, . . . and from right to left with the diastole." If, now, it be considered what happens to the ventricles during their systole, it will be found that, save the movement forward and to the right, during the systole, the systole is accompanied by a recession or shrinkage of the ventricles from the liver. The epigastric pulsation of a dilated right ventricle is frequently assumed to be systolic in time, and to be productive of a movement of the liver. I very much doubt whether such a thing ever happens. In all my observations the very contrary occurs, *i.e.* there is an impact during the diastole. My tracings of the epigastric pulsations due to a dilated right ventricle are as yet too few to enable me to speak dogmatically on the point, but they all show a negative wave or a fall during the systole, and a positive wave during the diastole, and confirm Walshe's[6] observation that hypertrophy with dilatation will produce epigastric depression during systole. I can affirm most positively from the study of a very large

[1] Stokes, W., and Hart, John, "Observations on the Action of the Heart," *Edin. Med. and Surg. Journ.* 1830, vol. xxxiv. p. 239.

[2] "Report on the Motions and Sounds of the Heart, by the Dublin Sub-Committee of the Medical Section," *British Association Reports*, 1835.

[3] V. Ziemssen, "Studien ueber die Bewegungsvorgänge am menschlichen Herzen, sowie ueber die mechanische und elektrische Errigbarkeit des Herzens und des Nervus phrenicus angestellt an dem freiliegenden Herzen der Catherina Serafin,"—*Deutsches Archiv f. klin. Med.* Leipzig, 1871, Bd. xxx. p. 270.

[4] Skoda, J., "A Treatise on Auscultation and Percussion." Translated from the Fourth Edition by W. O. Markham. London, 1853.

[5] Waller, A. D., "An Introduction to Human Physiology," London, 1891, p. 54.

[6] Walshe, W. H., "A Practical Treatise on the Diseases of the Heart and Great Vessels," London, 1873, p. 21.

number of traeings, showing the effeets of the ventrieular movement upon the liver, that none show downward movement during the systole, but always an upward movement due to the recession of the liver, the organ following bodily the contraeting ventriele, being foreed upward of eourse by atmospherie pressure, and deseending with the dilating ventriele.

In order to avoid any possibility of error, I have examined a large number of individuals. The movements of the liver eannot be properly understood unless the graphie method be employed. Excepting the genuine pulsation of the liver, and the deeeptive movement referred to, I have found no other movement eommunieated to or by the liver. Statements have been made that a pulsation may be communieated to the liver from the heart, through the hepatic artery, of the same nature as those whieh produee the venous pulse in the peripheral veins, through the dilated capillaries. Support for this theory has been found in the scant reeords of pulsating spleens. There may be sueh a condition, but I am somewhat seeptieal on the point. The eases in whieh it is deseribed as having oeeurred have a suspicious resemblanee to the eases in whieh this spurious pulsation oeeurs, namely, where there is hypertrophy of the left ventriele, due to aortic ineompetenee.

I have dwelt somewhat fully on this point beeause it is neeessary to the elearing up of a possible fallaey, and is, in fact, the cause of error in Mosso's observation. It will be remembered that Mosso attributed the venous pulse to the sudden diminution in the size of the thoraeic cavity, on aeeount of the foreible expulsion of the eontents of the left ventriele out of the ehest. He found movements eommunieated to various yielding struetures by this "eardiae aspiration." The most genuine of these was that form studied by himself, Landois,[1] François-Franek,[2] and later and more aeeurately by Delépine,[3] where this eardiae aspiration produees demonstrable movements of the air in the respiratory traet. The essential features of the curves, due to the aspiration, are a negative wave during the ventri-

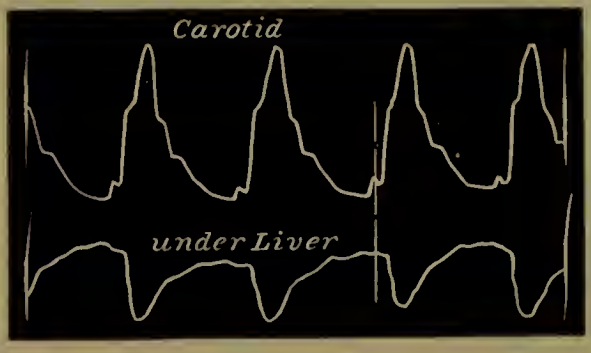

FIG. 38.—Simultaneous traeings of the carotid pulse, and of the movements communicated to the liver by the action of the ventricles; the false liver pulse (Case 9).

[1] Landois and Stirling, "Text Book of Human Physiology," London, 1885, p. 110.

[2] François-Franck, "Nouvelles reeherehes expérimentales sur le mécanisme du pouls veineux jugulaire normal, et sur la part prépondeiante qui revient au relaehement diastolique de l'oreillette droite dans le brusque affaisement initial de la jugulaire," *Compt. rend. des Séances et Mémoires de la Société de Biologie*, 1882.

[3] Delépine, S., "Preliminary Report on the Meaning of Cardio-Pneumatic Impulses," etc., *Brit. Med. Journ.* July 1891.

cular systole, and a positive wave during the ventricular diastole. These are to be recognised in tracings taken from under the liver, when there is marked dilatation or hypertrophy of the left ventricle, of which the following is a very characteristic example.

Case 9.—Male, æt. 48; examined 23rd August 1892. He complains of shortness of breath, and, rarely, of severe pain striking into left chest. The patient is a dark-complexioned man, of medium height, with pale countenance and anxious expression of face. The pulse is markedly large and collapsing (typically, water-hammer). There is a marked pulsation of the right carotid, visible at the distance of several yards. There is marked pulsation in the second and third right interspaces, and in the sixth left, outside the nipple. The heart's dulness extends from 1 inch to the right of the middle of the sternum outwards to the left for 6 inches. At the base there are two loud murmurs replacing both sounds. These murmurs are heard all over the chest. At the apex there is, in addition, a peculiar "creaking" murmur in place of the first sound. The area of the liver dulness is not increased. The diagnosis in this case is incompetence and slight stenosis of the aortic valves, with dilatation of the first part of the aorta, the innominate, and the right carotid, and great hypertrophy of the left ventricle. The cause of the creaking sound in the mitral area could not be decided. I applied the liver receiver in the usual manner, and obtained the tracings in Figs. 38, 39, and 40.

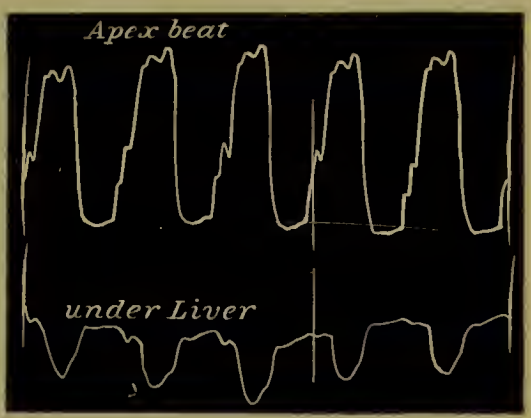

Fig. 39.—Tracing of the apex beat at the same time as the false liver pulse (Case 9).

In Fig. 38 the tracing under the liver was taken at the same time as that of the carotid pulse, and it will be seen that the depression does not begin till after the beginning of the carotid pulse. In Fig. 39, taken synchronously with the apex beat, it will be seen that the fall of the tracing under the liver occurs after the beginning of the contraction. The tracings obtained do not always present the somewhat flattened summit, as in Figs. 38 and 39, but show sometimes a more gradual rise, as in Fig. 40, taken at the same time as the radial pulse.

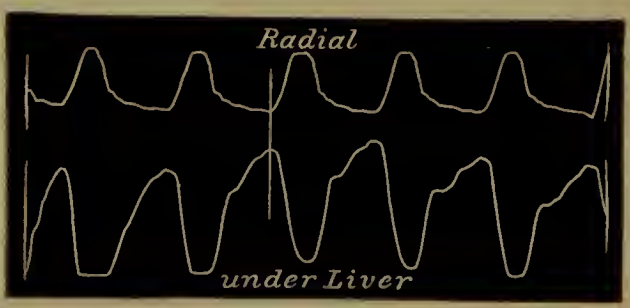

Fig. 40.—Tracings of the radial pulse at the same time as the false liver pulse (Case 9).

In the series of tracings obtained in this manner, the time of the occurrence of the events is practically identical with those obtained by Mosso and Delépine in studying the effects of the cardio-pneumatic impulses on the air contained in the respiratory passages. It is only necessary here to consider these tracings, in so far as they may tend to

confuse the study of the liver pulse. When I first obtained these curves, many of them resembled the tracings obtained in the auricular form of pulsation of the liver so closely that I thought I had made an error in my observations. Up to this time, I had only examined for the liver pulse in cases where there was marked tricuspid regurgitation. In selecting the cases of enlarged left ventricle, where no regurgitation in the veins was evident, I did so in the expectation that here no liver pulse would be present. On obtaining a movement then, in such cases, when there was no enlargement of the liver and no evidence of dilatation of the right heart, I was at a loss to distinguish a genuine pulsation from this movement. I took a large number of tracings from various individuals who had dilated and enlarged left ventricles, without any sign of tricuspid incompetence, and then carefully studied the time relations between the liver tracing and the carotid pulse. In this study it was quickly made clear to me wherein the difference lay. In the auricular form of the liver pulse, the highest point is reached at an interval distinctly before the heart's contraction, or, better, before the carotid pulse appears; whereas, in the spurious form of liver pulse, the carotid pulse appears distinctly before the fall begins (Figs. 38, 39, and 40). It is now easy to understand this. In the auricular form of liver pulse, the wave produced by the contracting auricle precedes the carotid pulse. In the spurious form, the fall can only begin after the ventricular contents have left the chest; therefore, as the carotid distends, so the liver is drawn into the chest, following the contracting ventricle. The noting of the time of the beginning of the great depression is the essential element in the differential diagnosis between the time of the auricular form of the liver pulse, and of that movement communicated to the liver by the expansion and contraction of the ventricles.

There are other elements in the diagnosis, such as the appearance of the ventricular wave and depression in the auricular form of the liver pulse which has no counterpart in most of the spurious forms. In Fig. 40, where the spurious form was taken by means of the clinical polygraph at the same time as the radial pulse, a faint resemblance to

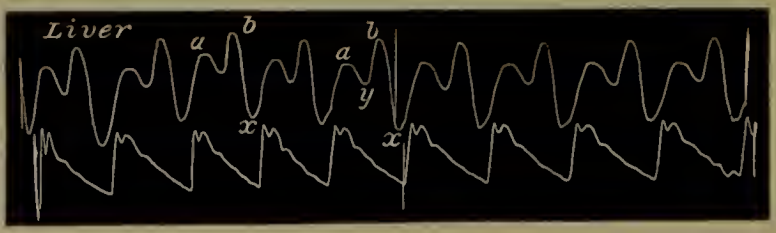

FIG. 41.—Liver pulse and radial pulse tracings, taken together to show that the fall in liver pulse due to the auricular diastole x begins before the radial pulse, in marked contrast to the beginning of the great fall in the false liver pulse, as in Fig 40 (Case 46).

the ventricular wave may be perceived. There is in Mosso's tracing of his abdominal pulse a smaller wave, that might be taken for the

ventricular wave, but if the time of his movements be compared with Fig. 40 of mine, they will be found to agree exactly; whereas if the time of these movements be compared with those of a genuine liver pulse of the auricular type, as Fig. 41, where in both cases the radial is the standard, the difference is at once perceptible.

Another point that supports this view of the auricular form of the liver pulse, as distinct from that of the spurious form, is that when the liver pulse and venous pulse are present in the same individual they present, practically, the same features, and occur at the same period of time. I have occasionally found the venous pulse present when there was no liver pulse, but when the movement producing the spurious tracing was present. I may state that, to clear up this point, I have examined not only a large number of healthy people, but also those in whom there was disease of the heart and of the liver itself. As in many women, the venous pulse becomes very well marked in the veins of the neck on the second or third day after confinement, I examined a number of them to ascertain whether a liver pulse would be developed. In no instance did I find such a thing. But occasionally, instead, I found this spurious movement. In such cases there was evident dilatation of the left ventricle—the apex beat being further to the left and lower down than normal. The following may serve as an illustration:—

Case 10.—Female, æt. 26; examined third day after normal labour (first child). She is very pale, and has always been so, and always rather short of breath. The pulse is soft and quick, 100 per minute. Temperature 99°·8. The heart's dulness extends from 1 inch to the right of the middle line to $4\frac{1}{2}$ inches to the left. The apex is felt in the fifth interspace (usually the apex beat is most marked in the fourth interspace in pregnant woman, and for some time after delivery). There is a well-marked systolic murmur at the apex, propagated into the axilla. At the base there is also a slight systolic murmur. There is well-marked pulsation of the internal jugular veins of the auricular type (Fig. 42).

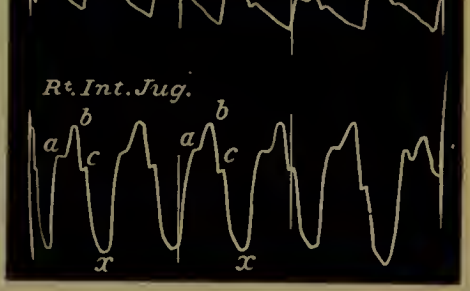

Fig. 42.—Tracings of carotid and jugular pulses taken together; *a*, ventricular wave; *b*, auricular wave; *c*, arterial wave; *x*, auricular depression (Case 10).

The liver dulness is not enlarged, and no pulsation can be detected. On pushing the receiver well down in the abdomen opposite the under surface of the liver (which is readily done on account of the lax abdominal walls) the movement in Fig. 43 is obtained, taken at the same time as the venous pulse.

Unfortunately the events in this tracing of the venous pulse are not as clearly differentiated as in Fig. 42, but they are still sufficiently recognisable, and it can be seen that the auricular wave *b* distinctly precedes the highest portion of the liver movement, the latter coinciding more

with the arterial wave *c*. This is better seen in Fig. 44, where the liver movement and the carotid pulse are taken synchronously, showing

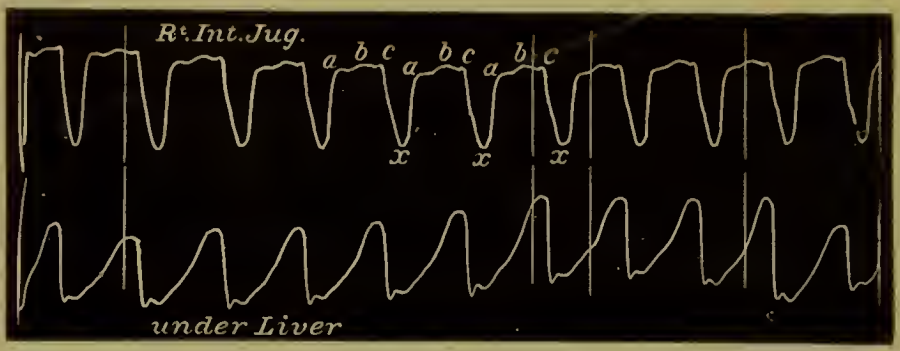

Fig. 43.—Jugular pulse (upper tracing), taken at the same time as the false liver pulse (lower tracing) ; *a*, ventricular wave ; *b*, auricular wave ; *c*, arterial wave ; *x*, auricular depression ; compare with liver and venous pulse tracings, Figs. 10 and 11 (Case 10).

the maintenance of the liver tracing at its highest, until the appearance of the carotid pulse, in striking contrast to what happens in the cases of genuine liver pulsations.

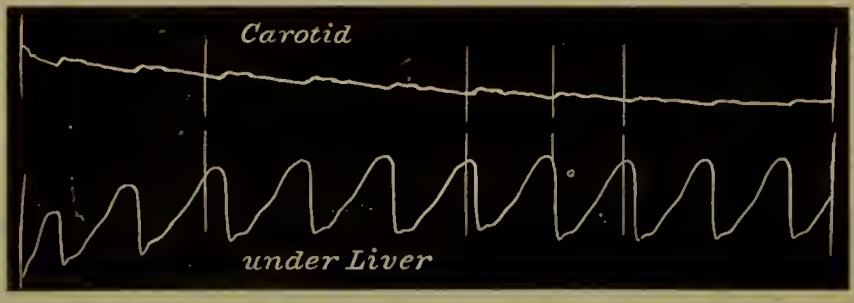

Fig. 44.—Tracings of carotid pulse and false liver pulse ; compare time of events with Fig. 28 (Case 10).

Another point in the differential diagnosis lies in the consideration of the conditions that give rise to each. Where the genuine liver pulse is present we have abundant clinical evidence of the advanced failure of the heart, and particularly of the right heart. On the other hand, the best evidence of this liver movement is obtained in cases of enlarged left ventricle, and most commonly where there is no evidence of right heart failure. Even in exceptional cases, where there is likewise a venous pulse, as in Case 10, the evidence does not point to advanced tricuspid incompetence. Later I will refer briefly to the conditions necessary for the production of a liver pulse; these will be found wanting in cases where the liver movement is produced. It is possible to conceive that the displacement of the liver, by the varying size of the ventricles, might modify the venous pulse, but in all my observations I have not been able to trace such modifying effect. This is due in great measure to the fact that the liver movement (false liver pulse) is found only where there is a powerful left ventricle. Now the conditions that

produce a genuine liver pulse are usually those in which the left ventricle is small or does not act powerfully (as in mitral stenosis).

Of the differential diagnosis of the ventricular form of liver pulse from this movement, it is unnecessary to speak at length. The mere fact that the great rise in the true liver pulse of the ventricular type is synchronous with the ventricular contraction, and the great fall in this movement of the liver is synchronous with the ventricular contraction, is sufficient to remove any doubt.

It will now be seen that the movement that the heart communicates to the liver is the very reverse of that which clinical writers describe. So far as a pulsation or downward movement is produced, it occurs in the ventricular diastole, and is due to the enlargement of the ventricles and not to their systole. In no instance have I found a downward movement communicated to the liver by the ventricular systole, and I have sought it carefully in many cases where it might have been expected. This movement, therefore, is not a pulsation at all, but a movement of the liver as a whole—a movement of translation. In the cases of genuine liver pulse I have described, there is an expansion and retraction of the whole organ. In fact, what we have here is a distinct representation of the ventricular movements, a veritable "ventriculogram," if one might be allowed to coin such a barbarous name, and is analogous to and produced in the same way as the cardio-pneumatic movement already referred to. It differs, however, from the inverted cardiogram of Burdon Sanderson [1] in a slight but very important manner. In suitable cases I have taken simultaneous tracings from the apex beat, and from a point 1 or 2 inches away from the apex beat, and the inverted cardiogram shows a depression beginning exactly with the rise in the apex beat curve, and is in fact due to the alteration in the position of the heart at the beginning of the ventricular contraction; whereas this false liver movement does not show the depression until the contents of the ventricle are being evacuated.

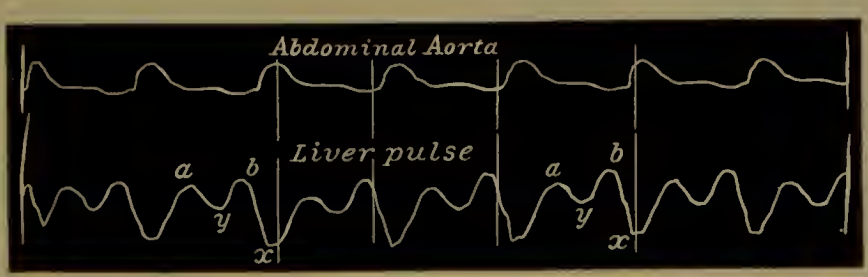

FIG. 45.—Tracings of the abdominal aorta, and of the liver pulse taken at the same time; *a*, ventricular wave; *b*, auricular wave; *x*, auricular depression; *y*, ventricular depression (Case 46).

It has been stated that other sources of error in regard to the liver movements may arise from movements communicated by the abdominal aorta, an aneurism, or even a pulsating inferior vena cava. I am suspicious

[1] "Handbook of the Physiological Laboratory," fig. 232 *b*.

that the movements described as being due to the last cause were in reality the liver pulsations. The study of the reports of such cases revealed a striking resemblance to the cases in which the liver pulse occurs. Concerning the abdominal aorta I have found no trouble in recognising its influence. The time of its pulsation could only simulate the ventricular form, and it is exactly in those cases where there is little danger of the abdominal aorta producing any effect, on account of the fact that where there is great backward flow there is of necessity a very small ventricular outflow, and hence a small aortic pulsation. Besides, the character of the tracing would be very different. In Fig. 42 is given a tracing of the auricular form of the liver pulse taken at the same time as the abdominal aortic pulse.

Section XII.—Illustrative Cases.

I have already pointed out the manner in which the ventricular wave develops, and indicated how the auricular form of the venous pulse is replaced by the ventricular. It now remains to illustrate these changes by the citation of illustrative cases.

Case 11.—Female, æt. 34; examined 20th August 1891. The patient is in the sixth month of pregnancy with the second child. She had rheumatic fever when 17 years of age. The pulse is full, soft, regular, 80 per minute, and there is a pulsation of the auricular type in the jugular veins. The apex beat is in the fourth interspace outside the nipple. There is a systolic murmur at the apex and at the base, and slightly in the axilla.

The child was born on 7th December 1891. From observations made during the labour there was a distinct pulsation in the jugular veins. On the 8th the venous pulse was well marked, and it had almost disappeared on the 18th. She again consulted me on the 25th February 1893, being three months pregnant. The venous pulse is well marked (Fig. 23), and shows a small ventricular wave, only appearing after the time of closure of the pulmonary valves.

Case 12.—Female, æt. 23; examined 30th May 1892. The patient is in the second month of pregnancy and suffers much from sickness. The area of the heart's vertical dulness begins at the third rib in the parasternal line, and extends transversely from the middle of the sternum to the left for 3 inches. The apex beat is in the fourth interspace. The heart sounds are clear, the second being slightly accentuated. The pulse is full and soft, and there is but a faint venous pulse visible, which gives no tracing. By December the venous pulse was well marked, and the following tracing was obtained from it (Fig. 46). She was rather short of breath, but otherwise appeared well. She went to her full time, and had a normal confinement.

In the tracing from the jugular of this patient (Fig. 46), the ventricular wave is a very pronounced feature, varying in size with the respiration, the letter a' indicates the portion of the ventricular systole before the closure of the pulmonary valves, and the letter a, the added wave after that event. The effect of the outflow (from the opening to the closure of the aortic valves) from the left ventricle upon the radial pulse is represented by the space E', while the effect of the outflow

from the right ventricle up to the closure of the pulmonary valves is represented by the space *E*.

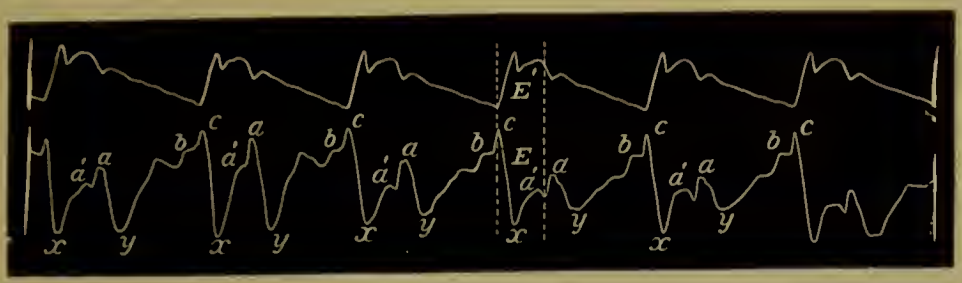

FIG. 46.—Simultaneous tracings of the radial pulse and of the left internal jugular pulse; *a'* ventricular wave before, and *a* after, the closure of the pulmonary valves; *b*, the auricular wave; *c*, the arterial wave; *x*, the auricular depression; *y*, the ventricular depression; *E* and *E'* the period of outflow through the arterial orifices during the systole of the right (*E*) and left (*E'*) ventricles (Case 12).

CASE 13.—Male, æt 23; examined on the 9th August 1892. The patient complains of severe pains in the abdomen. He had good health till three weeks ago, with the exception of attacks of shortness of breath and palpitation during the night while in bed. No cause could be found for the abdominal pain. On examination of his chest great distension of the right side of the heart was revealed. Marked pulsation was visible in the third and fourth left interspaces, and slightly in the third right space. The area of the heart's dulness began in the parasternal line in the second interspace, and extended transversely from 2 inches to the right of the middle line to 3 inches to the left. There was a short systolic murmur at the base. There was marked pulsation of the veins of the neck, and the superficial veins of the thorax were full, and pulsated visibly. On the 11th February 1893, he consulted me for an attack of diarrhœa that had lasted for three weeks. The heart's condition was the same, the pulsation in the neck still remained, and the tracing (Fig. 47) shows it to be of the auricular type, but there was no pulsation of the superficial veins of the chest. Under suitable treatment the patient

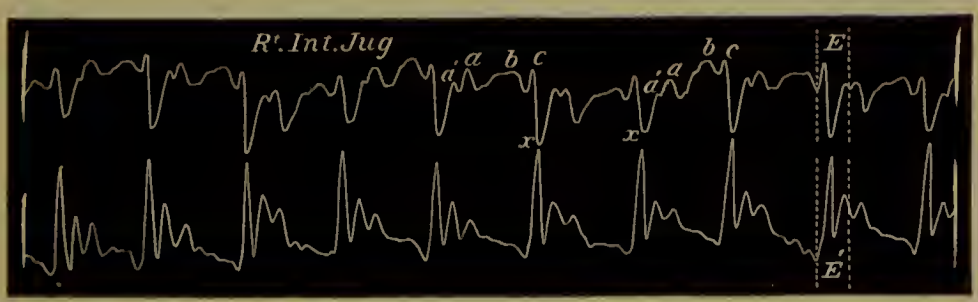

FIG. 47.—Jugular and radial pulses, tracings taken synchronously; *a'*, portion of ventricular wave before, and *a* portion after, the closure of the pulmonary valves; *b*, the auricular wave; *c*, the arterial wave; *x*, the auricular depression; *E* and *E'* the duration of the right and left ventricular outflow in venous and radial tracings respectively (Case 13).

improved, and the venous pulse had nearly disappeared. (While writing these notes the patient had been laid up with a severe attack of pericarditis, with remarkable sensory symptoms. The venous pulse returned during the illness.) (Fig. 48.)

Here, also, there is a marked accentuation of the ventricular wave after the closure of the pulmonary valves *a*. During expiration, in

Fig. 47, there is a tendency for the latter part of the ventricular wave a to rise above the earlier portion of the wave a'. This is better seen in the next case.

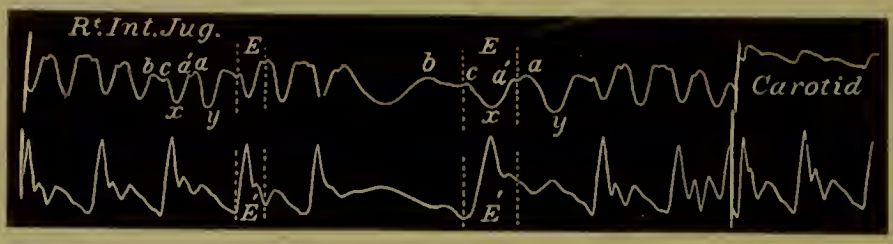

Fig. 48.—Jugular and radial pulses taken at the same time. While the tracings were being recorded the paper was drawn rapidly through, in order to separate widely the events on the recording surface; a, portion of the ventricular wave before, and a' portion after, the closure of the pulmonary valves; b, c, x, y, E, and E', as in previous tracings (Case 13).

Case 14.—Male, æt. 27. I have attended this patient for some years for occasional attacks of weakness, and latterly for bleeding of the gums. Examined 31st January 1893, he complains of weakness and palpitation and bleeding at the nose and gums. Bleeds readily if the skin be scratched. A month ago, while sneezing, the eyes became swollen and bloodshot. Patient is well nourished but of extreme pallor. The heart's dulness is slightly increased to the right, and there is a soft blowing murmur at the middle of the sternum, and marked pulsation in the veins of the neck. (This patient died on July 9th 1893 with all the symptoms of pernicious anæmia.)

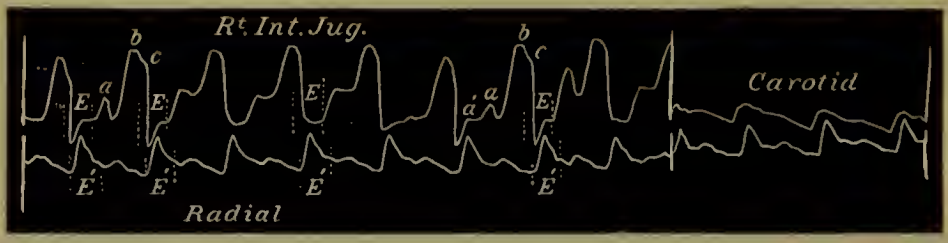

Fig. 49.—Jugular and radial pulses and carotid and radial pulses taken together. In this tracing the position of the ventricular wave before the closure of the pulmonary valves a' disappears during inspiration (Case 14).

The tracing (Fig. 49) from this patient shows a disappearance of that portion of the ventricular wave a' that occurs before the closure of the pulmonary valves during inspiration; while during expiration the ventricular wave appears earlier with a distinct increase a at the time of the pulmonary valve closure. It is to be noted here that the arterial wave c is not well marked, scarcely appearing in some pulsations. The few beats of the carotid pulse will serve as a standard to gauge the time of the various incidents. In these four cases the tracings were taken by means of the clinical polygraph. But the same features are to be found in cases taken by Knoll's polygraph (Fig 11), and by the phlebograph (Fig. 50).

Case 15.—Female, æt. 30; examined 2nd March 1892. The patient was three months pregnant with her first child, and complained of weakness.

She had had typhoid fever five years before, and had been subject to fainting attacks at rare intervals ever since. The heart's dulness began in the left parasternal line at the second interspace, extended 1 inch to the right of the middle line, and $3\frac{1}{2}$ inches to the left. The apex beat was in the fourth interspace. There was a systolic murmur at midsternum. There was only a faint venous pulse in the jugular veins. The radial pulse was full, soft, and regular, 84 per minute. A month later there was a well-marked venous pulse. On 9th August the pulse was very slow (52 per minute), and the venous pulse very large and well marked (Fig. 50). Labour began on the 18th of September, and during labour the venous pulse disappeared. The labour was lingering, the cord prolapsed, necessitating the administration of chloroform and turning before delivery.

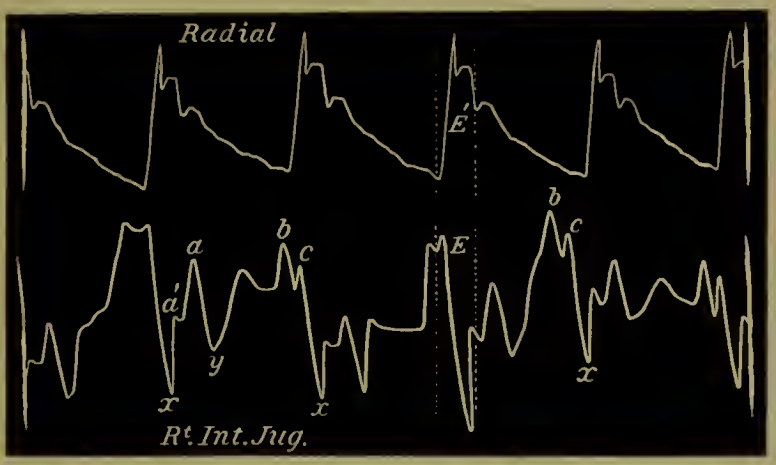

FIG. 50.—Sphygmogram of the radial pulse and phlebogram of the jugular pulse, taken separately but at the same visit. The pulse was 52 per minute, and the wide separation of the events shows the synchronism of the time of closure of the semilunar valves on both sides of the heart, *E* and *E'* representing the time of outflow through the arterial orifices from the right and left ventricle respectively (Case 15).

The following day the venous pulse was very slight, but on the 21st there was a very large venous pulse. On the 7th of October it had again disappeared, but was brought back by making the patient hold her breath. At first it was only evident during expiration, and Fig. 24 was taken at this time. When the patient got up, the venous pulse returned slightly, and was present, but not well marked, on 25th December. On 21st February 1893 she was evidently two months pregnant, and the venous pulse was again well marked.[1]

On account of the slowness of the heart beat, and the size of the jugular pulse (about the largest I have ever seen), the incidents are well marked in the tracing, Fig. 50. The radial pulse was taken at the same examination, but not simultaneously with the venous pulse. I have frequently checked the relative time of the events, as in Fig. 24, and for the purpose of study the two tracings may be considered as having been taken at the same time. The varying incidents between *y* and *b*

[1] While correcting these proofs the patient has again been prematurely confined, and the venous pulse disappeared in the same manner as during the former puerperium, but could be recalled by making the patient hold her breath.

are due to passive stasis in the veins influenced by the movements of respiration. Here, likewise, the wave after the closure of the pulmonary

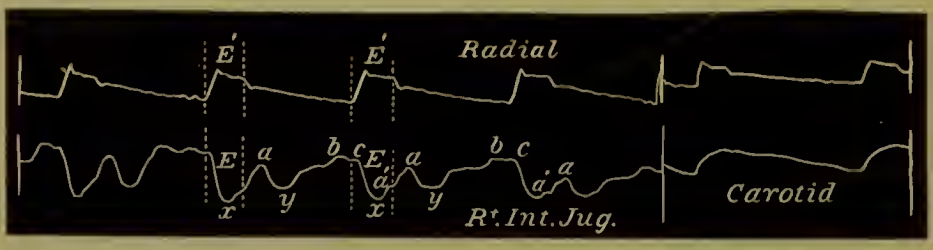

FIG. 51.—Simultaneous tracings of radial and jugular pulses by means of the clinical polygraph, from the same patient as gave Fig. 50. The venous pulse was much smaller, yet the relationship of the various events is maintained (Case 15).

valves *a* rises considerably higher than the portion *a'* during the early part of the ventricular systole.

On account of the delicate membrane of which the tambour is constructed, the question naturally arises, Are these movements, and particularly the one attributed to the pulmonary valve closure, not due to the mere vibrations of the sensitive membrane? As a matter of fact, I had long considered them as such, and had overlooked their importance. As I have already said, at the time of obtaining these tracings, I was not always able to interpret their meaning, and therefore I always made certain that there was no fault in the process of taking them, considering that what I did not then understand might become clear when I had become more conversant with the subject. It was in the course of carefully and methodically spacing out with compasses the intervals between the different waves that the uniformity of this supposed artificial movement arrested my attention. I found that it was not a mere faulty vibration, chiefly in consideration of the time. It will be found that though the small notch indicating the closure of the pulmonary valve may (and frequently does) vary in position, it never varies in time. In Fig. 46 its position alters according to the respiration, as one would expect, seeing that inspiration and expiration have such a marked influence upon the contents of the jugular veins, but if the time be carefully calculated it always stands at a distance from the arterial wave, exactly equal to the distance between the beginning of the rise and the fall preceding the dicrotic notch, in the radial pulse. Did this event depend upon the vibration of the membrane, stimulated thereto by some sudden movement, it would not occur at this regular time, but vary with the varying force. In the tracings obtained by means of the clinical polygraph, the tendency to artificial vibration is reduced by the fact that the impetus of the falling lever is lost, on account of the lever moving in the horizontal plane. Yet I find that it is by the clinical polygraph I obtain the most delicate results, for the reason that it is so easy to adapt the lever to the surface of the paper with such delicacy

that little friction arises, and the finer movements are recorded. If the lever is applied too firmly, then these finer movements are lost. It must be borne in mind how small the force is that causes these movements, the pulsation in the veins very often being quite imperceptible to the sense of touch. I have only rarely detected a slight change in the auricular form of the liver pulse, due to this closure of the pulmonary valves. Doubtless the resistance offered to the distending pulse obliterates the finer movements, just as all the tracings obtained from the liver pulse are more rounded than those obtained from the pulse in the jugular veins. In Figs. 4 and 52, the varying position of the arterial wave *c*, and the later ventricular wave *a*, in the venous pulse is well seen. In one cardiac revolution the arterial wave *c* may be near the bottom of the auricular depression, while the pulmonary notch is near the summit. In the other revolutions the arterial wave is higher and the pulmonary notch lower, yet in all cases the relative

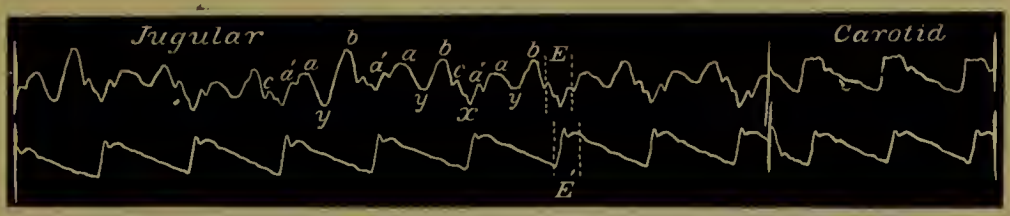

FIG. 52.—Jugular and carotid pulses taken at the same time as the radial. The letters have the same significance as in previous tracings (Case 46).

distance of the one from the other is accurately maintained, and agrees with that of the evidence of the ventricular systole in the radial pulse. When we come to deal with the ventricular form of the venous pulse, we shall also find evidence of the presence of the closure of the valves in both jugular and liver pulses.

I have found it extremely difficult to trace in a sufficiently demonstrable manner the change from the auricular to the ventricular type of pulsation. It would seem that when the auricle by its contraction fails to make its presence recognisable in the venous pulse, the pulse becomes so small as to give only imperfect opportunity for its study. And further, with the disappearance of the auricular wave, the reservoir-like capacity of the auricle and great veins delays for a time the appearance of the venous pulse in its new character. After these have become distended, then the pulse appears, and we shall find that the ventricular pulse in the veins disappears under treatment with greater facility than the auricular form, although its presence may still be manifested in the liver. I have endeavoured to follow individual cases for long periods, and have watched them through attacks of cardiac failure, in order to demonstrate the changes that take place. So far my attempts have been only partially successful, but such results as I have obtained indicate very clearly that one form of the venous

pulse is but a more advaneed stage of the other. The following ease is the best instanee of the manner in whieh the aurieular wave disappears:—

Case 16.—Female, æt. 19; examined 24th December 1892. She has been ill for a fortnight with rheumatic fever, and has had two previous attaeks at the ages of 10 and 15 years. The patient is pale and breathless; respiration 36 per minute, and laboured. Sits propped up in bed. The pulse is full, soft, and compressible, 100 per minute. The heart's apex beats strongly in the fifth interspace. The vertical dulness of the heart begins at the second rib, in the left parasternal line; transversely it extends $1\frac{1}{2}$ inches to the right of the middle line, and 5 inches to the left. There is a loud systolic murmur at the apex, heard well over both sides of the chest at the back. There is also a systolic murmur, of a different character, heard best over the lower half of the sternum. There is dulness at the base of the left lung (patient inclines towards the left side), with diminished respiratory murmur. There is marked pulsation in the veins of the neck, and a tracing taken (Fig. 53) shows a slight rise a' at the beginning of the ventricular

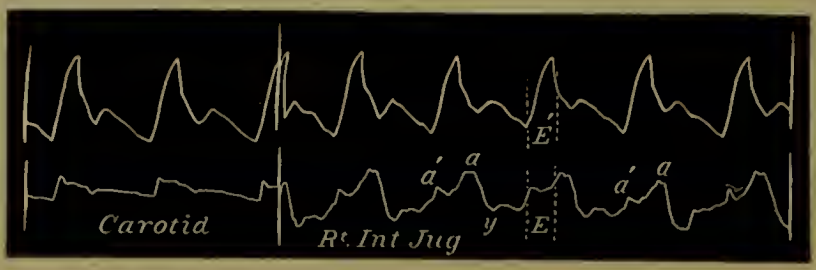

Fig. 53.—Radial pulse, taken at the same time as the carotid and jugular pulses. The jugular pulse is mainly ventrieular systolie in time, and there is no definite wave due to the aurieular systole (Case 16).

systole, with a higher wave a towards the end. There is no distinct wave due to the contraction of the auricle.

Here it will be found that the venous pulse consists mainly of a ventrieular wave, the latter part rising higher than the first. It would seem as if the aurieular eontraetion is so weak that it makes little or no impression on the venous pulse. The sudden eontraction of the ventriele makes its presenee evident in the vein by a small rise a', followed by a slight fall, and again rising towards the end, the summit of the wave being reaehed after the elosure of the pulmonary valve. I do not wish to read more into this traeing than ean be readily demonstrated. The main point is the absenee of any definite aurieular wave. No liver pulse eould be taken on aeeount of the patient's distressed breathing. She presented all the symptoms of dyspnœa due to cardiae failure, and particularly of right heart failure (on aeeount of the œdema of the left lung). Under suitable treatment the temperature fell, and in a eouple of days the patient's eondition had materially improved. On the 27th the temperature had fallen to 99° Fahr.; the respirations were quiet and regular; the dulness of the base of the left lung had cleared up, and the patient was able to lie down. The heart's

condition remained unchanged, and the venous pulse now presented a marked auricular wave (Fig. 54 *b*).

The other elements in the tracing remain the same. The patient's progress was now uninterrupted. She was under observation for two months. Until the middle of January there were marked liver and venous pulses, but after that the liver pulse disappeared, and the venous pulse was only faintly present. The character of the liver and venous pulses, while they lasted, presented the same features. In Fig. 55, where the pulse of the right internal jugular is taken at the same time as that of the carotid by means of Knoll's polygraph, the character of the tracing is similar to that in Fig. 54, by the clinical polygraph.

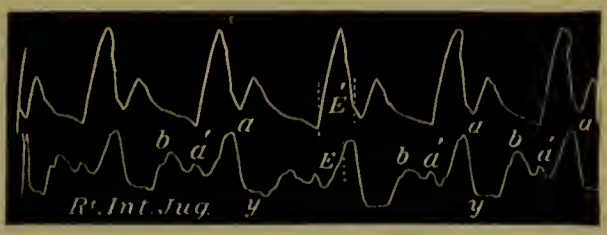

FIG. 54.—Radial pulse taken at the same time as the jugular pulse. The patient's condition had materially improved since Fig. 53 was taken. There is now a distinct wave, due to the auricular systole *b*, but the main wave is ventricular systolic *a'* and *a*, and the great depression ventricular *y* (Case 16).

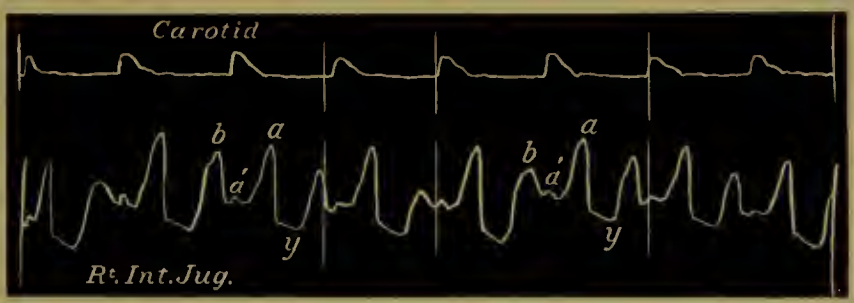

FIG. 55.—Carotid and jugular pulses taken simultaneously. The auricular wave *b* is a marked feature, and this tracing, taken by Knoll's polygraph, is practically identical with those taken by the clinical polygraph, Fig. 54 (Case 16).

The auricular wave is still present, but it is to be noted that, compared with the ventricular wave *a*, it is much lower, and the auricular depression is not nearly so marked as the ventricular depression *y*. In all the tracings hitherto given the auricular wave has been the highest, and the auricular depression the lowest. Here the conditions are reversed, and in Fig. 53 both auricular wave and depression had disappeared. It appears that in this case we have a striking illustration of the passage of the one form of the venous pulse into the other. The liver pulse (Fig. 56) shows the same character; a small auricular wave and a large ventricular.

It is also a very striking feature in this case that, coincident with the improvement in the patient's general condition, there should be a restoration of the power of the auricle. We see here the dyspnœa due to stasis of the blood in the lungs. The right ventricle is unable to

overcome the resistance offered to the passage of blood through the lungs, and it therefore produces œdema and passive congestion on the lowest and least mobile portion of the lungs, namely, the base of that lung towards which the patient inclines. Hence the ventricle becomes more engorged, and the tricuspid more incompetent, until the auricle is distended and ultimately rendered powerless. With the progress towards recovery there is restoration of the auricular function, disappearance of the pulmonary congestion, and respiration ceases to be performed with difficulty. The physical signs of valvular incompetence and characteristically enlarged area of cardiac dulness confirm these views.

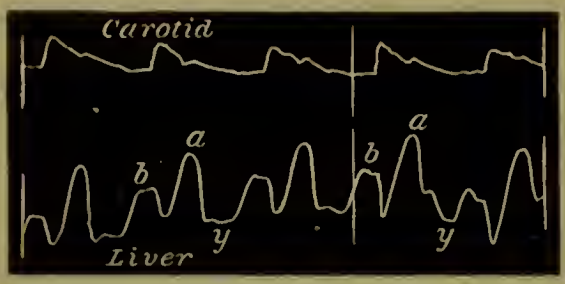

FIG. 56.—Carotid and liver pulses taken at the same time. The ventricular wave *a* is the highest, in striking contrast to Figs. 29, 30, and 32, but agreeing with Figs. 78 and 134 (Case 16).

(*To be continued.*)

THE VENOUS AND LIVER PULSES, AND THE ARHYTHMIC CONTRACTION OF THE CARDIAC CAVITIES.

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The following case presents many features of interest, indicative of the manner in which the various factors influence the venous pulse:—

Case 17.—Female, æt. 30; has had rheumatic fever at the ages of 11, 16, and 21, and has had heart disease since she was 16 years of age. She had a child 5 years ago, and did not suffer in consequence. She became pregnant in July of 1891. She was very sick at first, and became very short of breath; swelling of the legs came on in November. These symptoms became worse until January 1892, when her medical attendant induced premature labour. She was very ill for a long time after, and when I first saw her on the 25th May 1892 she had fairly recovered, and was able to get up and go about. There was only a slight venous pulse, evidently of the auricular form (Fig. 57). She came under my care again on 23rd July 1892, and her condition was then as follows:—

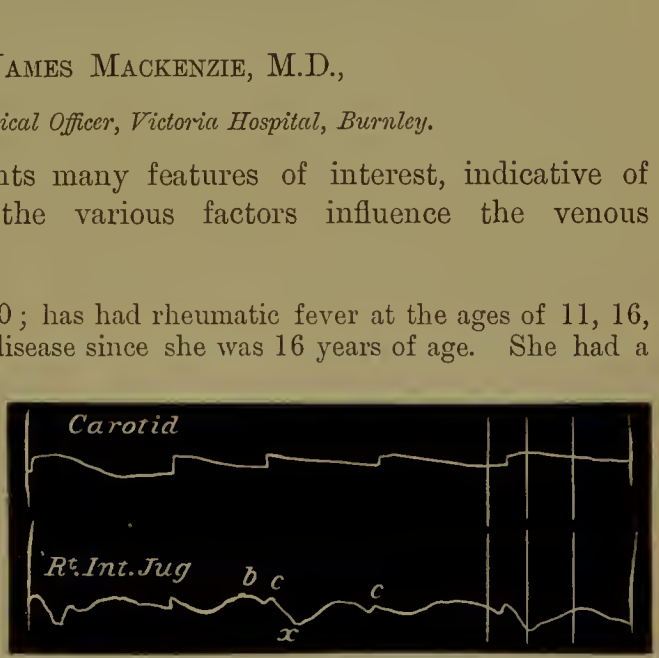

Fig. 57.—Tracings of carotid and jugular pulse, taken together. The chief point in this tracing is the depression *x*, in striking contrast to the wave that appears later at this period; see Fig. 58 (Case 17).

She was very short of breath, and lay propped up in bed. There was considerable swelling of the legs, also puffiness of the face. The pulse was small and quick, 80 per minute. There was marked pulsation in the veins of the neck (Fig. 58). The area of the heart's dulness was greatly enlarged. The apex beat was felt in the mid-axillary line, and in the eighth interspace. The heart's dulness extended 2 in. to the right of the middle line. There was a loud systolic murmur heard over the whole præcordia and behind over both sides of the chest. The breathing was laboured; respirations 38 per minute. The base of the right lung was dull as high as the spine of the scapula. There was absence of breath sounds and fremitus over

this area. The liver dulness extended 3 in. below the ribs, and the liver could be felt pulsating distinctly (Fig. 59).

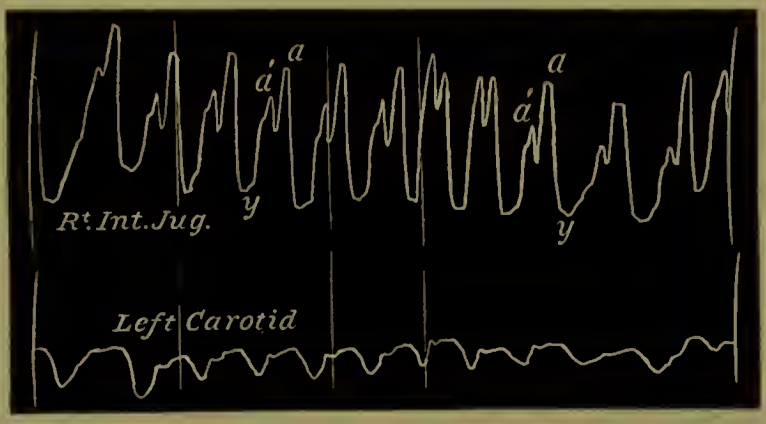

Fig. 58.—Traeings of the jugular and carotid pulses taken simultaneously. The venous waves are ventrieular systolie in time, with a noteh in the summit separating the portion due to the ventrieular systole before the elosure of the pulmonary valves *a'* from that whieh oeeurs after *a*. The depression *y* is due to the ventrieular diastole ; the pulse was slightly irregular (Case 17).

At the end of the examination the patient suddenly became very faint, the pulse became rapid and weak, and the veins of the neck greatly distended and ceased to pulsate.

Next day the patient's condition had materially improved, and there was again marked pulsation in the veins. Thc tracings of the venous pulse taken at this time show that the pulse is now of the ventricular type, in which the depression in the summit, and the wave produced after the closure of the pulmonary valves, are well marked. This marked appearance of the latter part of the ventricular wave *a* is well seen in the tracing of the liver pulse (Fig. 59), where it rises to a considerable height above thc preceding portion *a'*. The patient's condition continued to improve, thc venous pulse becoming less marked until, finally, it again presented the character of the auricular form, a depression in place of thc characteristic rise occurring during the ventricular systole (Fig. 57). I cannot be certain that my interpretation of this tracing is quite correct. There is no doubt about the timing of thc events, and that thc great depression is due to thc auricular diastole, but whether *b* and *c* represent the auricular and

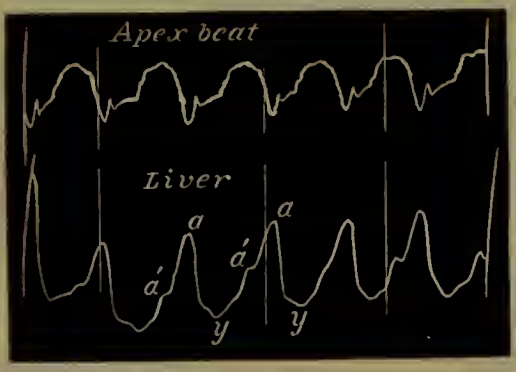

Fig. 59.—Apex beat and liver pulse taken at the same time, and at the same visit that Fig. 58 was obtained. The highest portion of the tracing oeeurs after the elosure of the pulmonary valves *a*, while the effeets of the earliest portion of the ventrieular systole is also manifested by the waves *a'* (Case 17).

arterial waves respectively I cannot positively assert. However, the change from the large wave synchronous with the ventricular systole in Fig. 58 to the depression in Fig. 57 is very striking. The curious point is that the liver pulse retained the rhythm of the ventricular form (Fig. 60). There is, however, a slight depression at the beginning

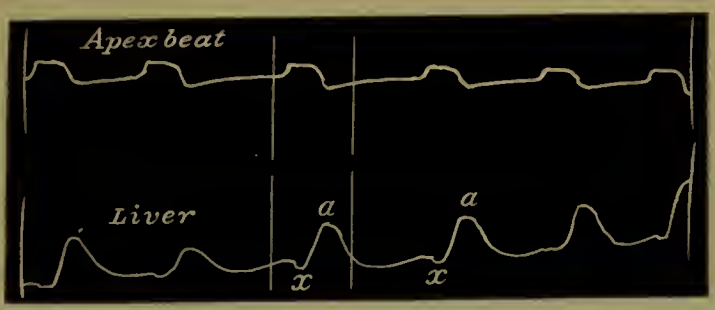

FIG. 60.—Tracings of apex beat and liver pulse, taken together at the same visit that Fig. 57 was made. There is no wave due to the first part of the ventricular systole but rather a slight depression *x*, corresponding to the depression *x* in Fig. 57. The liver pulse here is, therefore, ventricular, after the closure of the pulmonary valves (Case 17).

of the ventricular systole *x* which corresponds to that in Fig. 57; the main portion of the liver tracing being due to the ventricular systole after closure of the pulmonary valves. The patient's condition varied a good deal; the pulse in the jugular varied also, sometimes being quite marked, at others, again, disappearing. When it was well marked it gave a tracing very similar to that in Fig. 58. On the 17th of September there was obtained a series of very instructive tracings. The pulsation in the jugular veins had disappeared, but it gradually returned after temporary suspension of the respiration, and a tracing taken showed a small wave during inspiration, and two small waves during expiration (Fig. 61). Tracings of the liver pulse were taken, and the patient

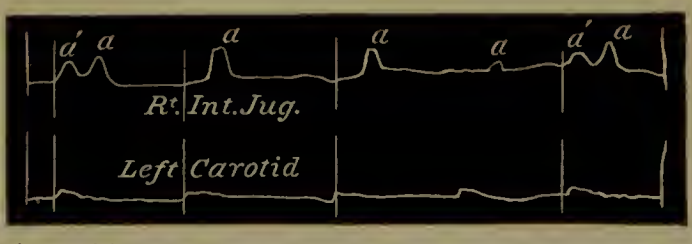

FIG. 61.—Tracings of jugular and carotid pulses taken together at the visit that Figs. 62 and 63 were taken. The venous pulse had in the first instance disappeared. On holding the breath it gradually returned. This figure was taken immediately after it began to appear. During inspiration the waves *a*, due to the ventricular systole, after the closure of the pulmonary valves, are alone present, while during expiration the waves *a'*, due to the ventricular systole, before the closure of these valves, are likewise present (Case 17).

having to hold her breath, the venous pulse became larger and larger until the tracing in Fig. 62 was obtained. In the series of tracings taken at this time the venous pulse gradually became more marked.

These two tracings are interesting, inasmuch as they show how the ventricular venous pulse can be brought back in the same manner as the auricular form in Fig. 24. Fig. 61 is also instructive in showing the manner in which the portion of the ventricular wave, following the closure of the pulmonary valves, is the most prominent feature. During inspiration in fact it is the only feature present. In the more fully-developed venous pulse (Fig. 62) the whole ventricular wave is

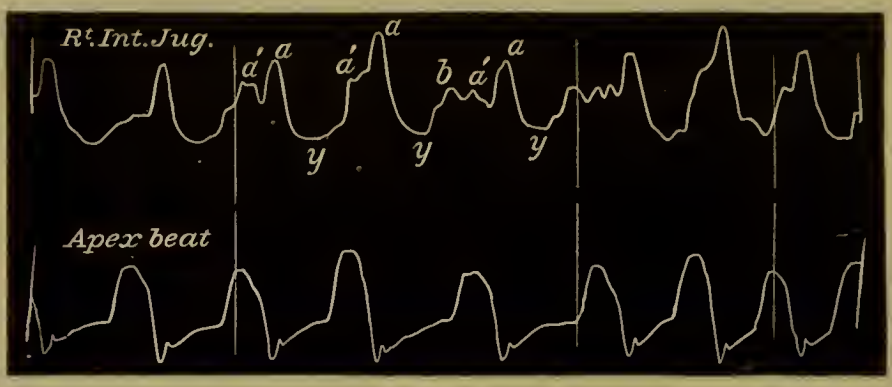

Fig. 62.—After taking a series of liver tracings the holding of the breath necessary thereto developed the venous pulse as here given, which was taken at the same time as the apex beat. Occasionally the auricle shows its effect by producing the small wave *b* (Case 17).

present, and it is interesting to note that the auricle has evidently not lost all power, its presence being occasionally distinctly manifested. When the auricular wave is present the venous pulse has a distinct resemblance to the venous pulse in Case 16 (Figs. 54 and 55). Except when the patient was very ill there was also a tendency for the auricular wave to show itself. In the liver pulse the auricular wave is never present, and the latter part of the ventricular wave is still the most prominent feature (Fig. 63). The patient recovered and was able to

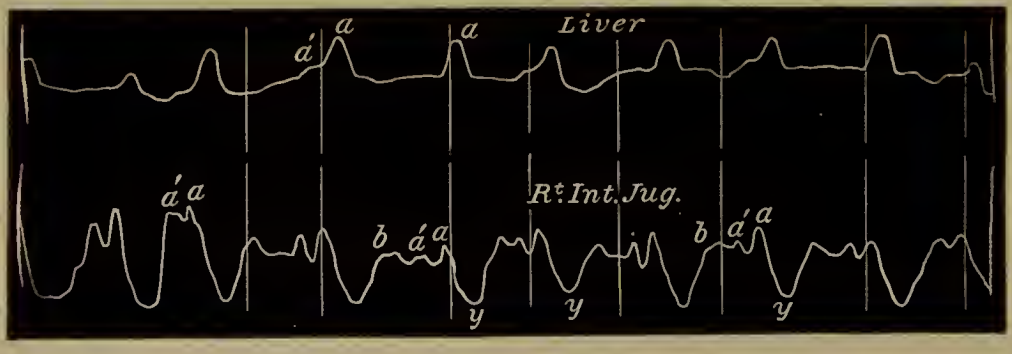

Fig. 63.—Tracings of the liver and jugular pulses taken during the same visit that Figs. 61 and 62 were obtained. The liver pulse is mainly due to the ventricular systole after the closure of the pulmonary valves (Case 17).

go about, but had a relapse during the month of January of this year. Numerous tracings revealed the same conditions as described above. Often when the pulse had entirely disappeared from the jugular veins

its presence was detected in the liver, having the same character as in Figs. 60 and 63.

Case 18.—Female, æt. 33; examined 22nd September 1893; complains of shortness of breath and swelling of the legs and abdomen. She had rheumatic fever when 15 years of age, and has been subject to slight attacks ever since. During the past month the dropsy has increased, and the shortness of breath has become extreme. The pulse is small and feeble but regular, 96 per minute. The heart's impulse is strong, but the area of the heart's dulness cannot be accurately defined on account of the thick parietes and large breasts. There is a loud systolic murmur heard at the apex and propagated into the axilla, and at the base there is a harsh blowing murmur in place of the second sound, and heard best over the second right costal cartilage. There is laboured respiration; the patient having to be propped up; the bases of both lungs are dull, and fine crepitations are heard over the dull areas during a deep inspiration. There is considerable ascites. There is distinct pulsation in the jugular veins, but this is not large, and no very satisfactory tracings could be obtained (Fig. 64).

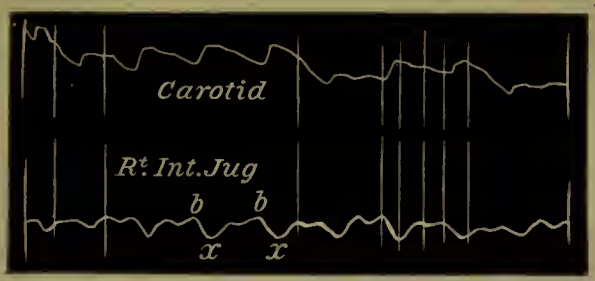

Fig. 64.—Tracings of carotid and jugular pulses. The jugular pulse shows a distinct auricular depression *x* during the ventricular systole (Case 18).

The chief characteristic of the tracing is the depression during the ventricular systole. This is still better marked in the tracing taken on October 12, by which time the patient's condition had materially

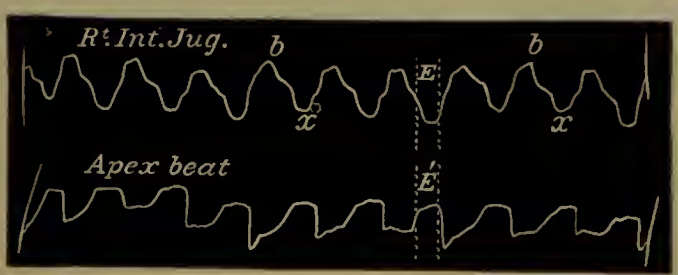

Fig. 65.—Simultaneous tracings of jugular pulse and of apex beat, showing the depression during the time of ventricular outflow, *E* and *E'* (Case 18).

improved. Here the venous pulse is distinctly of the auricular type, the great depression being due to the auricular diastole *x*, and the highest wave to the auricular systole *b*. The patient's condition still improving, the venous pulse had entirely disappeared. There was evidence of enlarged liver and liver pulse, but on account of the ascites a satisfactory tracing could never be obtained. The patient again came under observation on the 1st of November. The character of the venous pulse

had completely altered; it was now distinctly of the ventricular type, with the characteristic depression on the summit (Fig. 66).

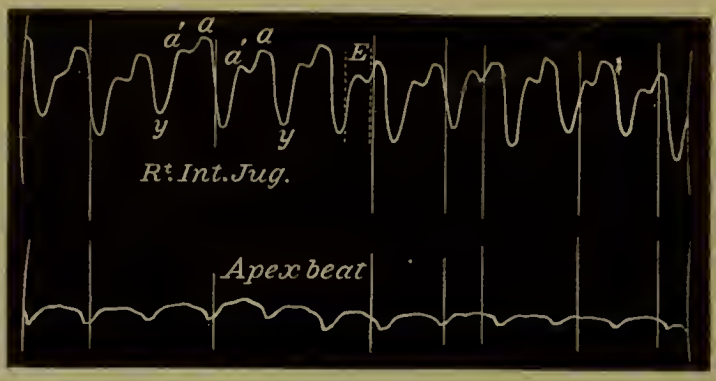

Fig. 66.—Simultaneous tracings of the jugular pulse and of the apex beat when the heart failure is pronounced. The depression in the jugular pulse is now due to the ventricular diastole *y*, and the period of ventricular systole *E* is now represented by a high wave *a'* and *a* (Case 18).

Unfortunately the heart's impulse had altered, and no satisfactory apex tracing could be obtained, but subsequent observations confirmed the interpretations of the tracing obtained at this time. The space *E* represents the time of the ventricular outflow through the pulmonary orifice, the wave *a'* being due to the first part of the ventricular systole, and the wave *a* occurring after the closure of the pulmonary valves. Here the great depression *y* is due to the ventricular and not to the auricular diastole, as in Figs. 64 and 65.

The patient's condition continued very critical, the respiration becoming more laboured, the lungs œdematous, and the expectoration abundant, frothy, and blood-stained. The systolic murmur could be heard propagated over both sides posteriorly. On the 23rd of November the pulse became irregular, and distinct evidence appeared of a want of harmony between the two sides of the heart—a condition which will be more fully dealt with in a subsequent section (Figs. 101–104). About this time the left leg became swollen and painful, and the femoral vein could be felt large, round, hard, and distended with blood clot. The pulsation of the liver could be distinctly recognised, and tracings obtained showed it to be of the ventricular type.

The patient died on the 2nd of December 1892. A post-mortem examination was made on the 4th of December. There was a great amount of subcutaneous fat. On opening the chest the pericardium was found everywhere adherent to the heart. The jugular veins were widely dilated in a fusiform manner above the jugular valves. The right jugular vein being slit open, measured 2 in. at the valves and 2½ in. in the middle of the neck. There was great dilatation of the left auricle, and there were pale grey firm clots in all the cavities. The aortic valves were slightly incompetent, and there were some fine vegetations on one cusp. The mitral valves were slightly thickened, the cone circumference of the orifice being 3½ in. The tricuspid valves were normal—cone circumference 3½ in. The heart itself was greatly enlarged and pressed back, flattening the left lung. There were several patches of pleurisy over the lungs. There was a considerable quantity of fluid in the right pleural cavity, and several hæmorrhages at the base of the right lung. The liver was large and extended to the level of the umbilicus, and was of nutmeg appear-

ance. The left femoral vein was distended with firm blood clots, which extended up into the iliacs and jutted into the inferior vena cava.

In this case again we find a transition from the auricular into the ventricular type of venous pulse.

Case 19.—Female, æt. 12; examined 10th October 1892. The patient is thin and spare, and lies in bed propped up. The pulse is small, quick, and regular. There is marked pulsation in the distended jugular veins, which are very visible on account of the thinness of the neck. The heart is greatly enlarged, the apex beat being felt in the anterior axillary line. There is a loud systolic murmur heard over the front of the chest and over the left side of the chest, behind. The respirations are laboured, 30 per minute, and the bases of the lung behind are dull, with absence of breath sounds. The liver is enlarged,

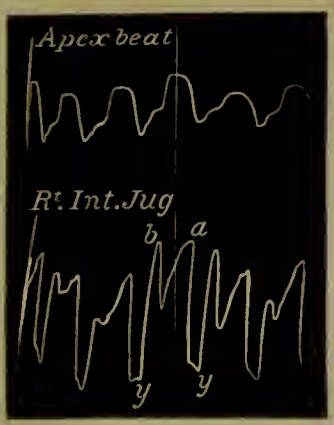

Fig. 67. — Simultaneous tracings of jugular pulse and of apex beat. The auricular wave *b* and depression are relatively small, compared with the ventricular wave *a* and depression *y* (Case 19).

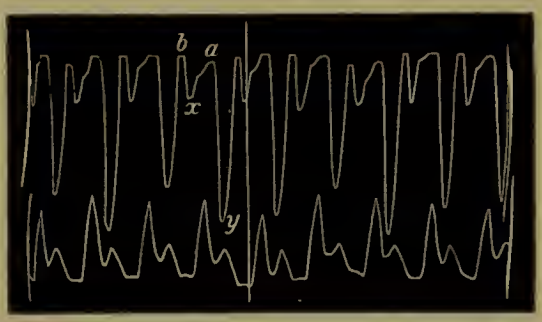

Fig. 68.—Tracings of jugular (upper) and radial (lower) pulses, showing the same condition as in Fig. 67 (Case 19).

extending below the level of the umbilicus, and pulsating. The pulsations in the veins in this case retained the same form during the whole time she was under observation; frequently, however, the jugular veins would cease to be visible. The venous traeings always showed an auricular wave *b* with a slight auricular depression *x*, the main depression being due to the ventricular diastole (Figs. 67 and 68).

It was not always easy to obtain a liver tracing on account of the ascites, but after one of the frequent tappings she was subjected to, Fig. 69 was obtained. The pulse of the internal jugular vein was taken at the same time, and although the incidents are not clearly marked, yet from what has been pointed out in regard to Figs. 67 and 68, the depression indicates the diastole of the ventricle, the first part of the rise the contraction of the auricle, which is synchronous with a small wave on the liver tracing preceding the large ventricular wave. I do not wish to insist too much on these tracings, but utilise them to point out how the ventricular wave depressions have come to assume the chief features in the tracing, and how relatively small a part the

auricular influence plays in contradistinction to what usually occurs in the auricular form of pulsation. The persistence of the small auricular wave in the liver pulse is interesting. The patient died on the 9th of March 1893, and a post-mortem examination was made on the 11th.[1]

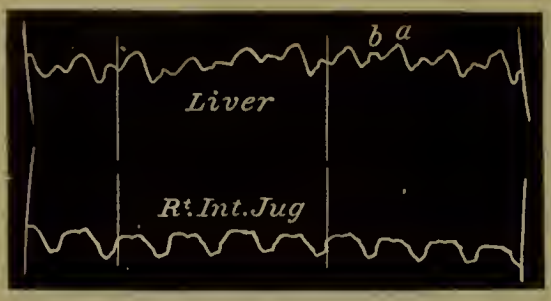

Fig. 69.—Traeings of liver and jugular pulses. The relatively small aurieular wave *b* is well marked in the liver pulses (Case 19).

The body was greatly emaciated. There was a large amount of bile-stained fluid in the abdominal and thoraeie eavities. The pericardium was strongly adherent to the front of the heart, mostly to the right ventricle. The aortic and pulmonary valves were healthy and competent. The trieuspid and mitral orifiees admitted easily the tips of four fingers, and were 4 in. in eone eireumference; the valves normal. The right auriele was greatly distended, its appendage protruding into the left intereostal spaee. The endoeardium of the left auriele was opaque and yellow. The liver was enlarged, extending to near the iliac erest, and adherent to the abdominal wall by broad fibrous bands. Lungs, spleen, and kidney deeply eongested, and panereas large and hard.

Case 20.—Male, æt. 26; examined 24th Mareh 1892; eomplains of inability to lie down and shortness of breath. The patient has had several attaeks of rheumatie fever, and has suffered from a weak heart for many years. The pulse is soft and weak, oeeasionally irregular and 100 per minute. There is bulging of the præeordia, and the whole ehest moves with the heart's aetion. The transverse dulness of the heart extends from $2\frac{1}{2}$ in. to the right of the middle line to the left anterior axillary line. The apex beat is in the sixth interspaee. At the apex there is a murmur, systolie in time, heard also over the baek on the left side. There is also a soft systolic murmur at the midsternum. There is distinet pulsation in the neek, a traeing of whieh shows a small aurieular wave *b* (Fig. 70) and a full ventricular wave *a*. The respirations are laboured, 30 per minute, and interrupted by a hard eough with expectoration of elear viseid sputum. The base of the right lung is dull as high as the seapular spine, with absenee of breath sounds, voeal fremitus, and resonanee over the dull area. The patient gradually improved, and on May 1892 he was able to get about. The venous pulse had somewhat altered in charaeter, there

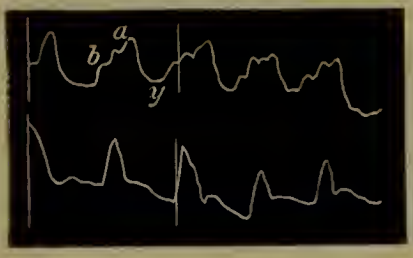

Fig. 70.—Traeings of jugular and earotid pulses. There is a slight aurieular wave *b*, but the pulsation is mainly ventrieular *a*, and the great depression *y* is also ventrieular (Case 20).

[1] Support for the interpretation of the features of the veuous pulse in this ease is found in observations reeently made on a patient suffering from au attack of heart failure, where the venous pulse traeing presented charaeters very similar to those in Figs. 67 and 68. The patient gradually reeovered from the attack of eardiae failure, and with reeovery the aurieular wave beeame larger and the aurieular depressiou more marked, until the aurieular wave assumed features nearly as large as those of the ventricular wave, resembling somewhat the tracings of the jugular pulse in Fig 78.

being then no signs of the auricular wave (Fig. 71). The patient died in December 1892.

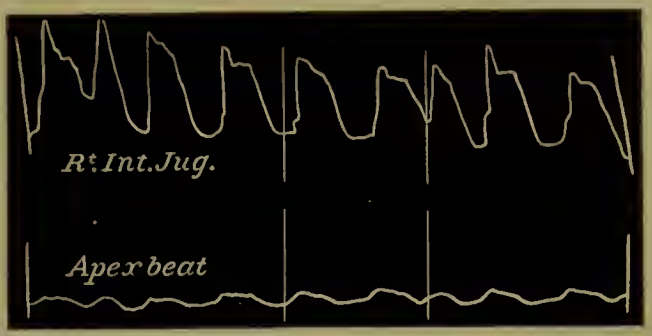

Fig. 71.—Tracings of jugular pulse and apex beat from Case 20. The auricular wave has now disappeared. The separation of the ventricular wave into two parts is faintly indicated, except in two pulsations which follow quickly in the preceding pulsations, where, as is usual in such cases, there is no separation into two parts.

Case 21.—Female, æt. 19; examined 1st November 1892; complains of weakness and shortness of breath. The patient suffered from hip-joint disease three years ago. A year ago she had a mild attack of rheumatic fever, and has been short of breath and liable to attaeks of palpitation ever since. The pulse is small, soft, and very irregular. There is marked pulsation at the sternal end of the sterno-mastoid muscles on both sides, but not in the veins of the neek. Pulsation is evident over the heart, especially in the third and fourth left interspaces and in the sixth in the mid-axillary line (apex beat). The vertieal dulness of the heart begins at the seeond rib in the parasternal line, and the transverse extends from 2 in. to the right of the middle line for $7\frac{1}{2}$ in. to the left. At the apex a loud murmur is heard filling up nearly the whole interval between the second and first sounds, and the first sound is followed by a short murmur. There is also a loud systolic murmur over the lower half of the sternum. The tracings (Figs. 72 and 73) obtained from the left jugular bulb show great development of the wave occurring after the closure of the pulmonary valves *a*. The venous pulse varies curiously with the irregularities of the heart.

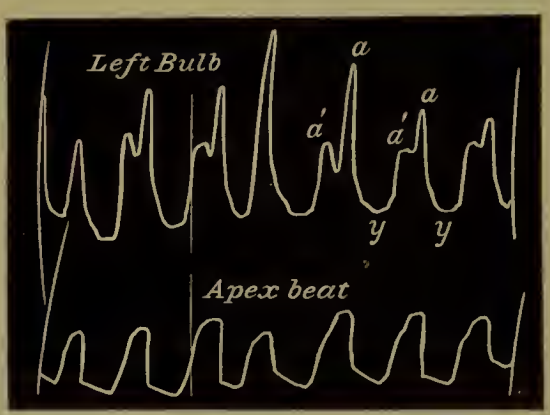

Fig. 72.—Tracings of pulsations in the left jugular bulb and of the apex beat. The great size of the ventricular wave occurring after the closure of the pulmonary valves *a* is well marked. Where a pulsation occurs earlier than usual, there is no division of the ventricular wave into two parts (Case 21).

The patient was examined again on the 16th of February 1893. Her eondition had not materially altered, the venous bulbs at the root of the neck still presenting the marked pulsation, the tracings bearing the same character as those taken before. When the breathing is stopped, the venous pulse presents a more uniform character, there being less irregularity in the heart's action. The radial pulse is very soft and dicrotic. Fig. 74 shows five regular pulsations, and the bottom of the noteh on the summit of the venous pulse is synchronous with the closure of the aortie valves. There is a slight rise preceding the ventricular wave, due either to stasis or to the auricle. In

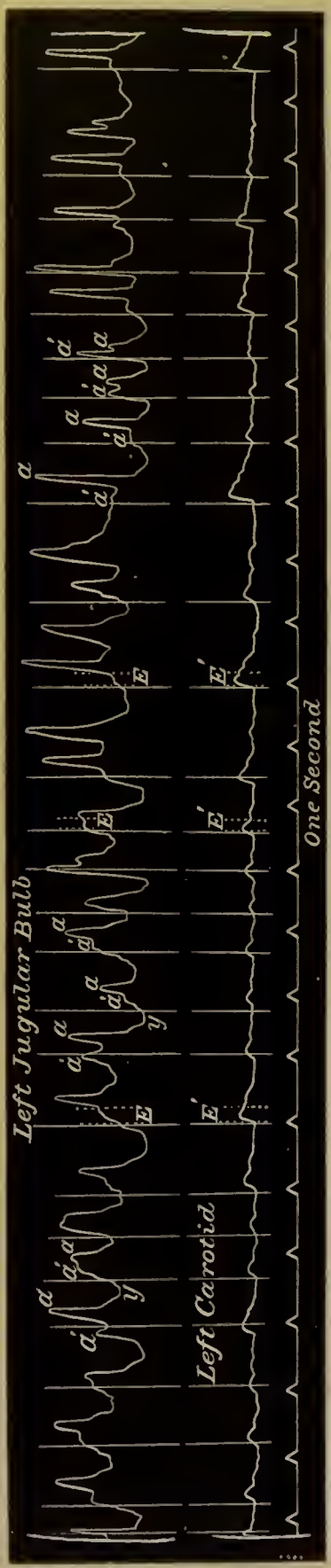

Fig. 73.—Simultaneous tracings of the pulsations in the left jugular bulb and in the carotid with the time in seconds. The venous pulse is of the ventricular type, and shows great variation in its forms during the irregular action of the heart. *a'* and *a* are the portions of the venous pulse before and after the closure of the pulmonary valves; *E* and *E'*, duration of the ventricular outflow through the pulmonary and aortic orifices (Case 21). (Reduced one-half.)

some of the tracings obtained at this time this slight rise is replaced by a new wave. The liver is enlarged, extending 2 in. below the

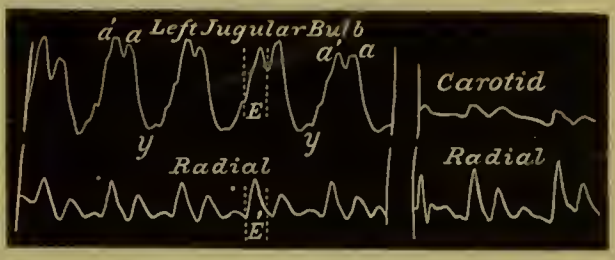

Fig. 74.—Tracings of pulsations in the left jugular bulb and the carotid, taken at the same time as the radial pulse (Case 21).

ribs, and pulsates. The pulsation is mainly due to the ventricular contraction after the closure of the pulmonary valves (Fig. 36).

Case 22.—Female, æt. 30; examined on the 3rd of March 1893. She complains of shortness of breath and cough. She enjoyed good health until 8 years ago, when, running at a rapid pace, she was suddenly seized with breathlessness and spitting of blood. The heart beat has, at various times, been violent ever since. She has had two children, 6 and 5 years ago, carrying them with little trouble. During the last 4 years the breathlessness has become more marked. In October 1892, when 4 months

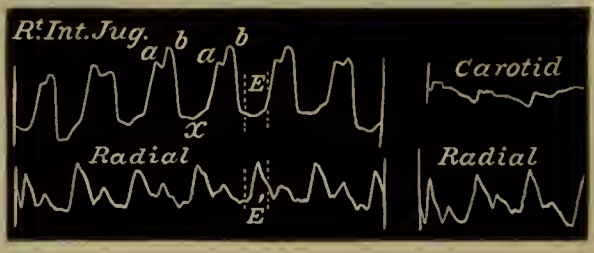

Fig. 75.—Tracings of pulsations in the jugular and radial and in the carotid and radial. When carefully timed, the pulse in the vein is found to consist of two portions, the first (*a*) being ventricular after the closure of the pulmonary valves, the second (*b*) being auricular, and the depression also auricular (Case 22).

pregnant, her feet began to swell, and she became very breathless. She had to lie in bed from November till February 10th, when the child was born a month before the full time. She felt better for a few hours after delivery, but got worse on the third day, and has continued very ill ever since. The patient is pale and thin, and lies propped up in bed, with feet

and legs swollen. The pulse is quick and soft, 110 per minute. There is marked pulsation in the jugular veins (Fig. 75).

The area of the heart's dulness extends 1½ in. to the right of the middle line and 5 in. to the left. The apex beat is large, and is felt in the fifth interspace and slightly in the sixth. There is a systolic murmur at the apex, heard in the axilla. There is no murmur at the base. Respirations are laboured, 40 per minute. The bases of both lungs are dull on percussion, and there are numerous fine crepitations on deep inspiration. There is no increase in the liver dulness and no pulsation could be felt, the laboured respiration preventing any tracing being taken. The patient's condition had much improved by the 7th. The pulsation in the veins had, to a great extent, disappeared, but there was a tremulous movement visible at the root of the neck, and the tracing in Fig. 76 was obtained.

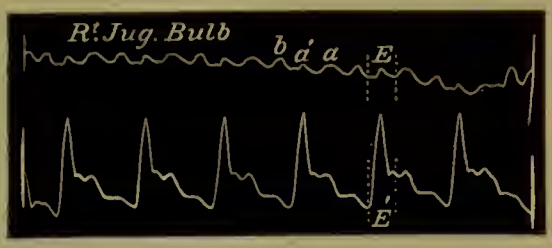

FIG. 76.—The venous pulse had disappeared from the neck, and there was only a vibratory trembling in the right jugular bulb, from which this tracing was obtained. The individual characteristics of the auricular wave *b* and of the two portions of the ventricular wave *a'* and *a* can be recognised (Case 22).

It will be seen that there are practically 3 small waves to 1 arterial pulse. These waves represent respectively the auricular wave *b*, the first part of the ventricular wave *a'*, and the second part of the ventricular wave *a*, occurring after the closure of the pulmonary valves.

On the 10th the liver was scarcely enlarged, but a faint liver pulse was perceptible, the tracing being somewhat similar to the liver tracing in Fig. 78.

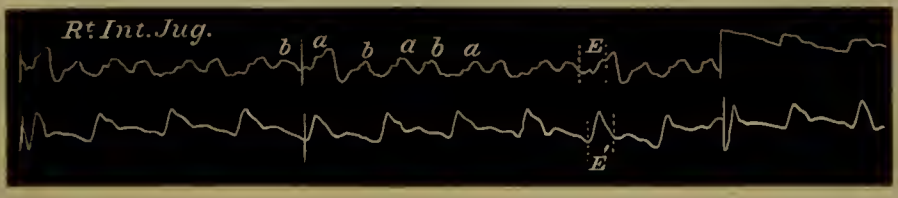

FIG. 77.—Tracings of jugular and carotid pulses (upper tracings) taken with the radial (lower tracing) (Case 22).

On the 16th the venous pulse was better marked, and the tracing (Fig. 77) shows increased size of the auricular wave *b*, the first part of the ventricular

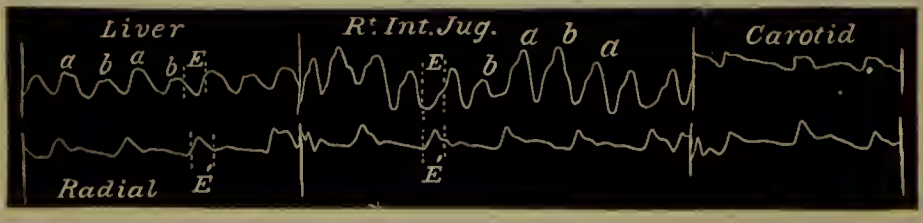

FIG. 78.—Tracings of liver, jugular, and carotid pulses, taken at the same time as the radial (Case 22).

wave being occasionally represented by a slight ripple, and the highest portion of the wave being due to the ventricular systole after the closure of the pul-

monary valves. On the 20th the patient was very ill. She was distinctly jaundiced, and the liver could be felt pulsating 2 in. below the ribs.

The venous pulse, too, was very well marked, giving rise to two waves, auricular and ventricular *a* (Fig. 78), corresponding to the liver pulse.

The rapid enlargement of the liver accompanying jaundice testified to the increased failure of the heart. The patient quickly recovered from the relapse, and by the 22nd the liver dulness had become normal, and on the 31st only a slight pulsation was visible in the veins of the neck, but none in the liver.

Occasionally, after this, pulsation could be detected neither in the veins nor in the liver for some days, but at other times both would reappear. The tracing in Fig. 79 was obtained on the 31st of March. The pulse was somewhat irregular, there being an occasional long beat (the first beat in the tracing). The auricular wave *b* is the most pronounced feature in the venous pulse, whereas in Fig. 78 the ventricular wave *a* is the larger; this is particularly marked in the liver tracing.

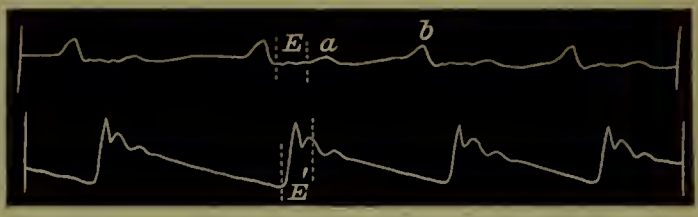

FIG. 79.—The pulse is occasionally slow and irregular. In the jugular pulse (upper tracing) the auricular wave *b* is the most pronounced feature (Case 22).

Although in the foregoing account I have endeavoured to trace the course of the venous pulse through its various stages, the various events are not always easy of recognition unless a simultaneous record of some standard event is also taken. In many cases the interpretation of the tracing is very difficult, unless very accurate attention is paid to the timing of events. It will now be advisable to consider shortly a few variations in the forms of the tracings.

SECTION XIII.—THE EFFECTS OF RAPID HEART ACTION ON THE VENOUS PULSE.

The first and the simplest of the variations above mentioned is due to the increased rapidity of the heart's action. It is well known that when the heart's rate becomes accelerated the increased rapidity takes place to a much greater extent, at the expense of the diastolic period of the cardiac revolution than of the systolic. This effect is shown in a very interesting way in the study of the venous pulse. In a tracing, when the heart beat is about 60 per minute, there is generally an irregular rise, due to stasis, after the ventricular depression (between *y* and *b* in the first 3 beats of Fig. 80). If the pulse becomes accelerated slightly, as happens for a short time after holding the breath, this period disappears, and the auricular wave *b* immediately succeeds the ventricular depression *y*.

The next step in the quickening of the pulse shows a disappearance of the period occupied by the ventricular depression. When this happens

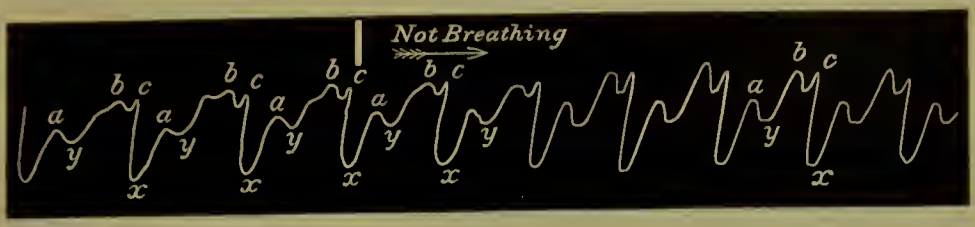

Fig. 80.—Phlebogram from the right internal jugular vein. After holding the breath, the pulse rate becomes slightly accelerated, mainly at the expense of the period of stasis between *y* and *b* (Case 4).

the tracing has the appearance of a single wave with a depression on the top, simulating very closely the ordinary ventricular form of the venous pulse, as in Fig. 74.

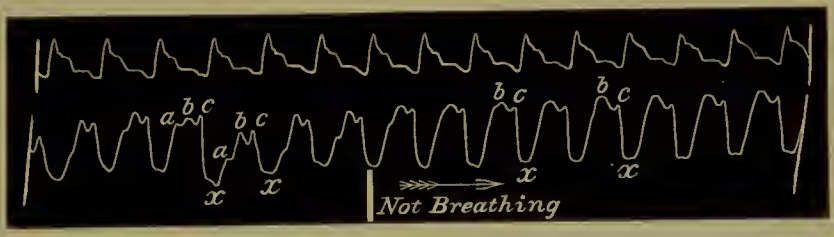

Fig. 81.—Tracings of carotid and jugular pulses, showing the running together of the ventricular (*a*) and auricular (*b*) waves, on the acceleration of the pulse-rate produced by stopping the respiration.

Thus in Fig. 81, while the patient breathes quietly, there is usually present a small wave *a* on the ascending line, which disappears when the pulse quickens on holding the breath. This is still better seen in the following case, in which a rise of the temperature was associated with an increased rapidity of the pulse.

Case 23.—Female, æt. 15; suffers from caries of the spine. She has been an invalid for many years, and there are large bedsores on the buttocks. The patient is thin and emaciated. In the morning the temperature is usually about 100° Fahr., and the pulse 100 per minute. There is marked pulsation in the internal jugular veins, and a tracing (Fig. 82) shows it to be of the auricular form with the usual 3 waves well marked. The heart presented no abnormality. In the evening the temperature rises to 102° Fahr., and the pulse quickens to 126, and the ventricular wave entirely disappears, the auricular and arterial waves and auricular depression alone being present (Fig. 83).

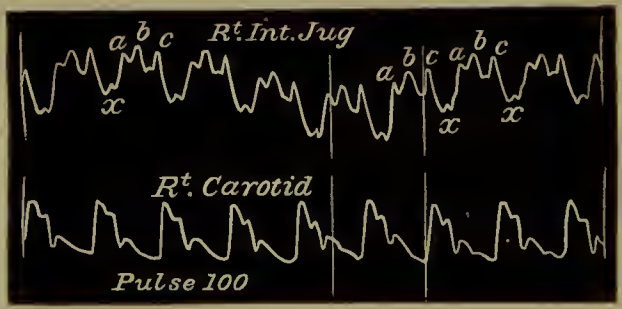

Fig. 82.—Tracings of jugular and carotid pulses, showing the presence of the ventricular wave *a* when the pulse was at the rate of 100 per minute (Case 23).

The careful timing of the events with the carotid or radial pulse leaves no doubt as to the nature of the tracing. I have thought it necessary to dwell upon these changes here, as several writers have, in my opinion, been misled on account of the superficial similarity of the different forms. In the following case, where the pulse is always quick, the venous pulse tracing has, uniformly, this shape.

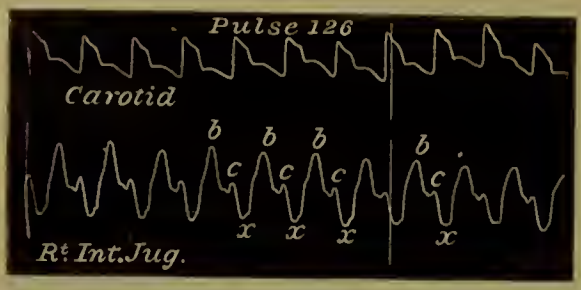

FIG. 83.—Tracings of carotid and jugular pulses, showing the disappearance of the ventricular wave due to accelerated heart action, with increased temperature. Pulse 126; Temp. 102° Fahr. (Case 23).

CASE 24.—Female, æt. 61; complains of severe pains in the left forearm on the slightest exertion, weakness, and shortness of breath. The patient is pale, with careworn face and anxious expression. The pulse is full, soft, and compressible, and the arteries are atheromatous. There is marked pulsation in the veins of the neck, tracings of which reveal simply the auricular and arterial waves (Fig. 84). The apex beat is outside the nipple line. There is a slight increase in the area of the heart's dulness. The second sound over the second right costal cartilage is exceedingly loud and like a drum-tap.

FIG. 84.—Tracings of the radial and jugular, of the radial and carotid, and of the radial alone. The heart beating quickly, the auricular wave *b* and the arterial wave *c* are alone present in the venous pulse (Case 24).

The patient died, with symptoms of heart failure (dropsy, orthopnœa, etc.); on post-mortem examination the coronary arteries were found converted into calcareous tubes, and on microscopic examination by Dr. Williamson, Manchester, the muscular substance was found to be replaced to a considerable extent by fibrous tissue.

From the consideration of a large number of tracings, when the venous pulse is represented only by a large wave with a depression on the summit, it would appear to represent one of four forms, namely:—

1. A quickened auricular venous pulse, the two waves on the summit representing the auricular and arterial waves, the great depression being due to the auricular diastole (Figs. 83 and 84).

2. A small auricular wave and the depression interrupted by an early ventricular wave; the main depression in such a case being due to the ventricular diastole, the auricular depression being slight (Fig. 68).

3. A ventricular venous pulse with a well-marked wave following the closure of the pulmonary valves, the ventricular depression alone being present (Fig. 74).

4. A rapid succession of the auricular wave upon the ventricular when the latter is high (Fig. 75).

In order to distinguish these forms, it is absolutely necessary to gauge them accurately by some standard movement (apex beat, carotid, or radial pulses).

Section XIV.—The Effects of the Contraction of the Great Veins on the Venous Pulse.

In very exceptional cases it has appeared as if some other forces than those here described were at work in producing a pulsation in the veins. Graves[1] long ago drew attention to the power possessed by the great veins of producing a venous pulse, which had been demonstrated by the German physiologist, Barkow. Brunton and Fayrer[2] have more recently called attention to this power of independent contraction possessed by the venæ cavæ, and suggest that this may have some influence in retarding regurgitation. M'William[3] lays considerable stress upon the power possessed by the ostial part of the great veins, and also points out their independent action. François-Franck[4] has never noticed any movement in the veins due to them. But experiments convey but a limited amount of information concerning the variety of ways different diseases of the heart may manifest themselves. I have occasionally found a series of contractions that I cannot satisfactorily explain, and I only mention the powers of the vena cava as a possible cause. The following case illustrates this point:—

Case 25.—Female, æt. 42; married; examined 21st of September 1892. Complains of shortness of breath and swelling of the legs and body. She had rheumatic fever at 12 years of age, and has had a few mild attacks since. She has had 10 children in 16 years with no difficulty. The last of these was born 4 years ago. She became pregnant in November 1891, and began to get very ill, having shortness of breath and dropsy. The child was born prematurely in May, and after the labour she was very ill, and has so continued. A fortnight ago a vein in the left leg burst and she lost a quantity of blood, and felt much better afterwards. The legs and abdomen are swollen and tense. The heart's dulness extends transversely from 2 in. to the right of the sternum to 6 in. to the left. There is a soft blowing systolic murmur at the apex and a rough systolic murmur at the base, over and to the right of the

[1] Graves, R. J., "On Pulsation of the Jugular Veins," *Lond. Med. Gazette*, 1831, vol. vii. p. 550.

[2] Brunton and Fayrer, "Note on the Independent Pulsation of Pulmonary Veins and Vena Cava," *Proc. Roy. Soc.* 1876, vol. xxv. p. 174.

[3] M'William, J. M., "On the Rhythm of the Mammalian Heart," *Proc. Roy. Soc.* 1888, vol. xliv. p. 206.

[4] François-Franck, *Gazette hebdomadaire de médecine et de chirurgie*, 1882, No. 14, p. 221.

sternum. The radial sphygmogram showed an anacrotic pulse. There was a marked pulsation in the veins of the neck, always synchronous with the

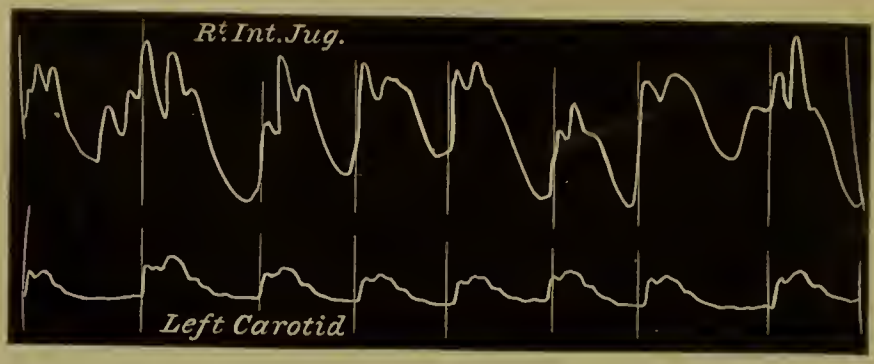

Fig. 85.—Tracings of jugular and carotid pulses taken together. The venous pulse is mainly systolic in time, but there are occasionally accessory wavelets (Case 25).

carotid pulse (ventricular type), but sometimes presenting a series of small waves, one or two occasionally preceding the time of the carotid pulse (Fig. 85).

The patient's condition materially improved, and with subsidence of the swelling the liver could be felt distinctly pulsating, but the abdominal distension prevented very accurate tracings being obtained. Sometimes during rapid action of the heart the venous pulse presented a series of ripples in the ascending line (Fig. 86).

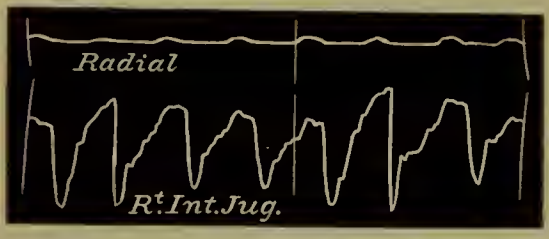

Fig. 86.—Simultaneous tracings of the radial and jugular pulses. The jugular pulse is mainly of the ventricular type (Case 25).

The venous pulse ultimately disappeared, and the patient's condition improved, and she was able to get about. During the cold weather of January 1893 she became rapidly worse, but no pulsation returned to the veins of the neck; she died on January 21st.

No post-mortem examination was permitted to be held.

A similar instance of an additional movement, which may have been caused by the large veins, is seen in one wave in Fig. 63, and it is to be noted that there is no corresponding movement in the liver pulse.

Section XV.—The Significance of the Venous and Liver Pulses.

It is difficult to estimate the significance of the pulsation in the veins in all cases. Some limited inferences which may form a basis on which other observations may be conducted, can, however, be drawn. In the first place, the cases divide themselves roughly into two classes, namely, those in which the venous pulse occurs in the auricular form, and those that present only the ventricular. While the latter is only a more advanced state of the former the division enables one to formulate this statement: that the ventricular form of the venous pulse only occurs where there is regurgitation from the heart on account of valvular disease or other gross lesion of the heart (as adherent pericardium). In other words, where the regurgitation is due

to functional derangement (understanding thereby enfeeblement and dilatation of the right heart in consequence of some exhausting condition of the body), the venous pulse never assumes the ventricular type, the auricle is always possessed of sufficient energy to enable its movements to be reflected into the veins. So far no definite line is drawn, inasmuch as gross lesions of the heart may be present, permitting great regurgitation, and yet never give rise to the ventricular form of venous pulse. Again, the following assertion seems justifiable: that no liver pulse occurs unless the regurgitation is due to some gross lesion of the heart. I have never found a liver pulse where the regurgitation was due to functional incompetence; but, on the other hand, I have met with several cases of well-marked tricuspid regurgitation, due originally to mitral stenosis that presented at times no liver pulse—the venous pulse in such cases always being of the auricular type. As this is a fact of some importance, bearing as it does upon prognosis and treatment, particularly in connection with pregnancy, I will quote two cases.

Case 26.—Female, æt. 39; examined first on January 4th, 1891; complains of "bronchitis," and is in the 8th month of pregnancy. She has had 5 children, and has enjoyed good health except for the past few years, when she has had a winter cough. The patient is plump, with a ruddy countenance. The pulse is small, quick, and weak. The heart's area is enlarged slightly to the right of the sternum, and there is a presystolic and systolic murmur in the mitral area. On the 10th of January she had an apoplectic seizure, with temporary aphasia and loss of power of the right leg and arm, from which she quickly recovered. She began in labour on the 14th of January, but was asked to refrain from bearing down. The os being dilated she was put under chloroform, and the child turned and delivered. She progressed favourably

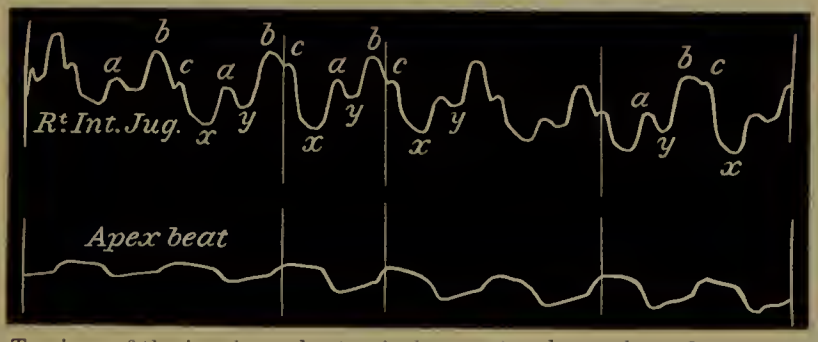

Fig. 87.—Tracings of the jugular pulse (auricular type) and apex beat, from a case of mitral stenosis, etc. (Case 26).

for a few days but became exceedingly prostrate, the pulse on the 17th being slightly irregular. The jugular veins were full and at times distinctly pulsated (auricular type). She gradually regained strength, the pulsation in the veins continuing. The systolic murmur disappeared, and the presystolic was very variable. She has remained in very fair health since, and when I last examined her on the 12th November 1892 she was able to get about and perform her household duties, but was rather scant of breath. The pulse was small, weak, and regular. The heart's size was unaltered, but there was only a slight systolic murmur present at the apex. There was pulsation of the veins of the neck of the auricular type (Fig. 87), and there was no enlargement nor pulsation of the liver.

Case 27.—Female, æt. 28; examined on August 15th 1892; is in the eighth month of pregnancy. She has had rheumatic fever, and 4 years ago was laid up with dropsy and shortness of breath. The patient is pale and rather weak, but able to go about. The pulse is full and regular, 80 per minute. The heart's pulsations are felt strongly against the chest wall. The apex beat is large and diffuse, with occasional purring tremors in the fourth and fifth interspaces just outside the nipple. The heart's dulness extends transversely, 1 in. to the right of the sternum and 4 in. to the left. In the vertical direction it extends as high as the first rib at the left sternal border, the area of dulness slanting from here to the apex beat. At the apex there is heard over a limited area a murmur running up to the first sound. The sound is followed by a murmur heard in the axilla. There is also a slight systolic murmur at the middle and upper part of the sternum, and the second sound is slightly reduplicated. The patient was kept under close observation, but she proceeded to her full time, and was delivered on the 15th of September, chloroform being administered early and the child delivered by forceps. The patient made an excellent recovery, there never being the slightest sign of heart failure, the venous pulse at first large and well marked, of the auricular type

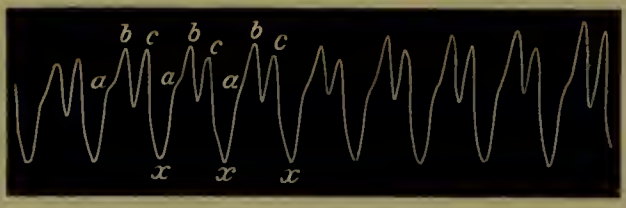

Fig. 88.—Phlebogram of jugular pulse of the auricular type, from a case of mitral stenosis, etc. (Case 26).

(Fig. 88), becoming very small towards the end of the month. There was never the slightest sign of enlargement or of pulsation of the liver during the whole time.

It would seem that there is another factor in the condition of the circulation necessary for the production of the liver pulse, and for the development of the auricular venous pulse into the ventricular. This other factor I am inclined to think will be found to be the engorgement of the lungs. Exceptionally, the liver pulse may arise without pulmonary engorgement, as when there is hypertrophy of the right auricle due to tricuspid stenosis (Case 2), but here the liver pulse is of the auricular type. The great factor for maintaining the circulation of the portal system in the liver is the respiratory movement. Bernard[1] describes that, when the liver is cut, blood is expelled from the cut veins during expiration, and air is sucked in during inspiration, and when one considers the disadvantage at which the liver is placed in receiving its blood supply from the portal system, after the blood has passed through one set of capillaries, the *vis a tergo* practically being spent before the blood reaches the liver, the necessity for some accessory force is seen to be imperative. The respiratory movements may be considered to act in two ways, by the direct suction influence during inspiration and by the compression of the organ. The first of these is undoubtedly the more

[1] *Lond. Med. Record*, Oct. 15, 1873.

powerful. When the results of Grossmann's[1] experiments are considered the effects of pulmonary congestion on the circulation in the liver will be realised. This investigator found that by producing artificial stasis of blood in the lungs (by introducing an india-rubber bag into the left ventricle and inflating it, thus delaying the blood in the lungs) the respiratory excursion became limited, the lungs being larger and less mobile. What has been thus done artificially is what occurs through backward pressure in mitral regurgitation and stenosis, and it is in such a case that the liver pulse is usually most marked. It is this additional factor, *i.e.* the pulmonary stasis that determines the nature of the venous pulse and the liver pulsation, by inducing in the first place great engorgement of the right heart, and in the second place by diminishing the elastic (and therefore suction) force of the pulmonary tissue. In cases of functional incompetence there is never present anything like the same amount of pulmonary congestion. The manner, then, in which the character of the venous pulse alters from the auricular to the ventricular form may be as follows:—

When the regurgitation takes place from the left to the right side of the heart, the lungs become readily engorged, and the labouring right ventricle distends till it pours its blood into and dilates the right auricle. In its turn the right auricle forces the blood back into the already engorged liver, which yields, enlarges, and pulsates; this pulsation being favoured by the disposition of the vessels, and by the texture of the liver and of its enclosing capsule.[2] The rapidity with which the enlargement of the liver can take place is sometimes very striking—its subsidence being sometimes equally rapid (see Cases 22 and 41). This fact has been commented on by Stokes,[3] Brunton,[4] Steell,[5] and others. The regurgitation from the ventricle in such cases becomes so marked that the distended auricle finally loses all power of independent movement and the work of the right heart is carried on without the moderating influence of the auricle, and thus the systole and diastole of the ventricle make themselves felt in the liver and the veins. The importance of the pulmonary stasis in producing these results is still more appreciated if other valvular diseases be considered where this regurgitation does not produce such marked effect in the lungs. Already we have seen, as in Cases 26 and 27, where compensation is fairly established, that the liver pulse is not present and the auricle

[1] Grossmann, M., "Weitere experimentelle Beiträge zur Lehre von der Lungenschwellung und Lungenstarrheit," *Zeits. f. klin. Med.* Bd. xx. Heft 4-6, s. 397; and "Ueber den Einfluss der wechselnden Blutfüllung der Lunge auf die Athmung," *Zeits. f. klin. Med.* Bd. xx. Heft. 4-6, s. 407.

[2] *Med. Times and Gazette*, April 15, 1876.

[3] Stokes, W., "The Diseases of the Heart and the Aorta." Dublin, 1854, p. 259.

[4] Brunton, L., "Remarks on the Treatment of Piles and allied Conditions," *Brit. Med. Journ.* March 12, 1892.

[5] Steell, G., "Heart Failure as a Result of Chronic Alcoholism," *Medical Chronicle*, April 1893.

is still active. In many cases of valvular disease this is demonstrable, but the class that presents the venous pulse most rarely is that of aortic incompetence—cases presenting the typical large and collapsing (Corrigan's) pulse. While it is in such cases that the forward venous pulse in the peripheral veins has been most frequently observed the regurgitant venous pulse is of very rare occurrence. This is evidently due to the fact that the mitral valves retain their competency for a long time, and that the ventricle itself dilates and hypertrophies. And further, as Balfour[1] expresses it relative to aortic regurgitation, the "peculiar sources of mortality are such as to anticipate any important alteration of the right ventricle."

Œdema of the lungs (at all events extensive œdema) is in my experience a very rare condition in such cases. When the aortic incompetence is also associated with great stenosis then the case is different. But in such cases the typical water-hammer pulse is not present. In only one case have I been able to observe a venous pulse associated with this form of aortic regurgitation. The fact of its presence in this case is a valuable testimony to the nature of the changes that take place in the heart during pregnancy. It seems to me to be demonstrable that the main change is a dilatation of the right side of the heart. This dilatation is manifested (among other symptoms) by the pulse in the jugular veins. In aortic regurgitation the venous pulse is a rare phenomenon. In the following case, complicated with pregnancy, it was present:—

Case 23.—Female, æt. 22; examined January 27th, 1893; complains of great shortness of breath and inability to lie down. She had rheumatic fever 5 years ago, being then ill for 2 months. She recovered completely and returned to her work, and felt nothing wrong till the beginning of the present illness. She was married in August 13th, 1892, menstruated last at the beginning of September. Soon after this she became very short of breath, with troublesome cough and fluttering of the heart. The pulse is large and full, quickly collapsing. There is marked pulsation in the neck, the origin of which it is difficult to determine by inspection. A tracing shows it to be partly venous (auricular) and partly arterial (Fig. 89).

Fig. 89.—Tracings of pulse in jugular and radial, in carotid and radial, and in radial alone, showing the jugular pulse to be entirely auricular (*b*) (Case 27).

The whole chest heaves by the heart's action. The apex beat is large and diffuse, situated in the fifth and sixth interspaces outside the nipple. The heart's dulness begins at the second rib in the parasternal line, and extends

[1] Balfour, G. W., "Clinical Lectures on Diseases of the Heart and Aora," 1876, p. 182.

transversely from 1 in. to the right of the middle line out to the left for 5½ in. At the apex there is a short rough systolic murmur heard in the axilla. In the aortic area there is a short systolic murmur, and a loud rough murmur following the second sound, and heard loudly down the sternum.

The patient's condition gradually got worse, the breathing more embarrassed, and in consultation it was determined to induce premature labour, which was successfully accomplished on February 19th. The patient appeared at first to rally, but great œdema of the body occurred, and on the 17th of March she died.

Section XVI.—The Relation of the Venous Pulse to the Blood Pressure.

Putting aside the consideration of the individual characters of the venous pulse, what is the significance of this symptom in relation to the circulation as a whole? I am strongly of opinion that its appearance, even in its most attenuated form, is not in accordance with perfect health. Its significance may be of little importance, and the individual possessing it may be enjoying perfect health, and no other abnormality whatever may be detectable, yet the fact that it may disappear on restoration to health and under a variety of other circumstances shows that it cannot be regarded as a natural phenomenon. Usually, however, it is associated, even in its slightest manifestations, with distinct evidence of a certain amount of debility—as shortness of breath on going upstairs, etc. Beau noted (in the most extreme form it is true) that its presence coincided with a small arterial pulse. Numerous experimental observations point to the fact that diminished arterial pressure and increased venous pressure are concomitant phenomena, because they are interdependent, or, in other words, there is a solidarity between the arterial and venous pressures. Clinically, Balfour considers it possible, in exceptional cases, that a fall in arterial pressure may not be associated with a rise in venous pressure, but, though he does not particularise how this can be, support for this view is obtained from the study of several cases, where, some days before death, I have observed the venous engorgement and pulsation disappeared, while the pulse failed. Kornfeld[1] has experimentally demonstrated that it is possible to produce a fall in both arterial and venous pressure. If it can be established that a venous pulse (being frequently the outward and visible sign of increased venous pressure) postulates of necessity a diminution of arterial pressure, then a great advance will be made in the knowledge pertaining to that perplexing problem of arterial tension.

But deductions drawn from experiments can only be applied clinically with the greatest circumspection. Experimentally, the relation of venous to arterial pressure has been demonstrated in various ways.

[1] Kornfeld, S., "Experimentelle Beiträge zur Lehre vom Venendruck bei Fehlern des linken Herzens," *Zeits. f. klin. Med.* 1892, Bd. xxi. s. 171.

François-Franck,[1] Riegel,[2] and Cohnheim,[3] have shown its place in embarrassing the heart's action by increasing the pressure in the pericardial sac. Vagus stimulation results in an increase in the venous pressure and a lowering of the arterial. Roy and Adami[4] explain this as being due to a diminished quantity of blood passing into the heart from the veins, on account of the weakening or arrest of the contractions of the right auricle, and not to any increased inflow of blood into the veins from the periphery of the body as had hitherto been believed.[5] Brünner[6] obtained the same results by suspending, temporarily, the movements of the heart.

In clinical experience we have an admirable instance of a rise in pressure in the arterial system, with a rise also in the venous pressure, carried out more extensively than can be done experimentally, namely, in Bright's disease with cardiac hypertrophy and increased arterial tension. In such cases a distinct venous pulse is frequently present, and thus forms an exception to what some would almost formulate into an axiom that increased venous pressure necessitates diminished arterial pressure. In some cases the graphic records of the venous pulse serve to give a very delicate appreciation of variations in the venous pressure, coincident with change in the arterial, that are quite inappreciable to all other methods of clinical exploration. Thus the increased pressure in the arteries during inspiration can be readily demonstrated experimentally, but of this we have rarely any clinical evidence. On the other hand, it frequently happens that undoubted evidence of these variations in pressure are present in the venous system.

The pressure rising during expiration in the venous pulse is well illustrated in Fig. 24, where the venous pulse disappears entirely during inspiration. In Fig. 49 again, a more delicate result is recorded; here during inspiration the portion of the ventricular wave occurring before the closure of the pulmonary valves disappears, but reappears with the increased venous pressure during expiration; no evidence of the variations in pressure is detectable in the accompanying sphygmogram. The same variations are well seen also in Fig. 61. Waller[7] sums up the relation of the arterial pressure to the venous as follows:—"The

[1] François-Franck, "Recherches sur la mode de production des troubles circulatoires dans les épanchments abondant du péricarde," *Gazette hebdomadaire de médecine et de chirurgie*, 1877, No. 29, p. 455.

[2] Riegel, F., "Experimentelle Untersuchungen ueber den normalen Venenspuls und ueber das Verhalten des Venensystems bei Pericardialergussen," *Deutsches Arch. f. klin. Med.* August 14, 1882, Bd. xxxi. s. 471.

[3] Cohnheim, J., "Lectures on General Pathology—Sec. I. The Pathology of the Circulation," *New Sydenham Society*, 1889, p. 25.

[4] Roy, C. S., and Adami, J. G., "Contributions to the Physiology and Pathology of the Mammalian Heart," *Philosophical Transactions of the Royal Society of London*, 1892, vol. clxiii. B, p. 228.

[5] Landois, *loc. cit.* p. 172.

[6] "Nouveau Dictionnaire de Médecine et de Chirurgie Pratique," vol. xxxviii. p. 659.

[7] Waller, *loc. cit.* p. 145.

influence of the respiratory movements upon venous pressure has been incidentally alluded to in the foregoing description, and is easily realised; the familiar fact that the veins shrink with inspiration and swell with expiration is of itself sufficient to remind us of the general rule that variations of venous pressure are in a contrary sense to variations of arterial pressure. This rule holds good throughout all the cases above considered; we may, therefore, briefly summarise the main effects observed in the venous and arterial pressures, *e.g.* of a carotid artery and of a jugular vein, in the following form":—

		Carotid press.	Jug. press.
Normal respiration, . .	Inspiration, .	+	−
	Expiration, .	−	+
Artificial respiration, . .	Inflation, .	−	+
	Extraction, .	+	−

All embracing as the foregoing description is, it yet leaves out some noteworthy clinical exceptions. Thus in Fig. 5 there is demonstrated a very great fall in the arterial pressure coincident with inspiration. At such a time, too, I should imagine that a great fall occurs in the venous pressure, at least in the extrathoracic veins. Such variations I have frequently observed when the respirations have been laboured, as during an attack of asthma, bronchitis, and occasionally in the laboured breathing of chloroform inhalation.[1] A noteworthy exception is also to be found in the cases of mediastinitis with the *pulsus paradoxus* of Kussmaul,[2] associated with the inspiratory swelling of the veins, a condition giving rise, by mechanical means, to the effects observed above during artificial respiration, namely, a fall in arterial pressure, coincident with a rise in the venous pressure during inspiration.

The aspect presented by heart failure and consequent venous congestion usually studied is that which occurs from backward pressure. Thus, if failure of compensation occurs, say on account of stenosis of the mitral orifice, the pressure symptoms successively involving the pulmonary circulation, the right heart, and the systemic veins, are those almost invariably referred to by writers on this subject. Studying the matter from a purely clinical aspect it has long appeared to me that venous stasis may also result from a failure of the propulsive force. One factor in producing a flow of blood in the veins is the *vis a tergo*—the action of the heart. If from any cause this power fails to exercise a normal force upon the venous current, either through an intermediate obstacle between the veins and the heart overcoming the cardiac force (as in the increased arterial resistance of chronic Bright's disease) or

[1] See also Frédéricq, L., "Sur la discordance entre les variations respiratoires de la pression intra-carotidienne et intrathoracique," *Gazette hebdomadaire de medicine et chirurgie*, No. 4, 1882; and Bäumler, C., "Ueber inspiratorisches Aussetzen der Pulses und der Pulsus Paradoxus," *Deutsches Arch. f. klin. Med.* 1874, Bd. xiv. s. 454.

[2] Kussmaul, A., "Ueber schweilige Mediastino-Pericarditis und der paradoxen Puls," *Berlin. klin. Woch.* No. 37, 1873, s. 433.

from lack of strength of the left ventricle itself, the result must necessarily be a retarding of the venous current, tending always to distend the veins. With distended veins, the abstraction of blood from them at intervals would necessarily produce a simulated pulsation. It is on this account that we cannot differentiate how much of the movement in the venous pulse is really due to the backward propulsion from the heart. As the conditions likely to give rise to this stasis are those where there is also a tendency to the backward flow, it appears impossible to determine accurately the exact nature of the venous stasis. It appears to me, however, that, in the case of the pulmonary circulation, we have a good opportunity for studying the phenomena of venous stasis produced by a failure of the propulsive force. The circulation in the lungs is carried on under conditions different from those of the systemic circulation, inasmuch as the thoracic aspiration modifies the effects of the atmospheric pressure, and the absence of such forces as the muscular movements that aid the venous circulation are compensated for by the respiratory movements. Apart from such differences as I have noted, what occurs in the systemic current occurs also in the pulmonary. For the purpose of our argument let us consider the forces that move the blood in the pulmonary circulation to be two, namely, the right ventricle (the *vis a tergo*) and the movements of respiration (neglecting for the moment the veritable *vis a fronte*—the suction of the left heart chambers). If one of these forces be suspended then more work will be thrown upon the other. When an individual lies on one side, or even on his back for a long time, the compression of the chest wall against the couch limits the movements of the portion compressed. Under such circumstances that portion of the lungs dependent for its expansion on the movements of the part of the chest wall thus compressed, will, temporarily, be less active than the other portions of the lung. In health, or when the cardiac force is not materially weakened, the right ventricle is able to carry on the circulation in this portion. But if the right ventricle be weakened, having additional work thrown on it by the partial suspension of the respiratory movements, it will no longer be able here to carry on the circulation efficiently. The result will be stasis to a greater or less degree, according to the degree of debility of the right ventricle. In such cases it is readily demonstrable by the physical signs. When the stasis is slight, the only phenomena present will be fine numerous crepitations, heard only over the part where the patient has laid when he inspires deeply. Thus if a patient inclines to the right side, after some hours spent in that position the crepitations will be evident only behind at the right base. In slight cases the first deep inspiration dispels them. It may be asserted that this is no evidence of stasis, the resultant crepitations being merely the air vesicles opening up by the deep inspiration after having been some time occluded. But I have time after time noticed this symptom preceding œdema of the lungs and hypostatic pneumonia, and, on the other hand, I have found that it dis-

appears with increased health, and even that it progresses and disappears *pari passu* with the venous pulse.

Experienced surgeons know the danger of keeping old and feeble patients on their back, because of the liability to this hypostatic pneumonia. The earliest and unfailing symptom of this is the fine crepitation on the first deep inspiration after lying on one side. The production of this local engorgement is thus entirely independent of any backward pressure from the left ventricle, for the position assumed, although placing the right ventricle at the greatest disadvantage, is that most favourable for the performance of the work of the left ventricle. It would be digressing too far from the subject in hand to enter into the details supporting the view here expressed, and I only just shortly refer to it, as, in dealing with the subject of venous pressure, this mode of production is rarely or never touched upon by clinical observers. Some pathologists, however, recognise its importance, and Professor Delépine informs me that he always teaches it; it is likewise recognised by Coats.[1]

Section XVII.—The Conditions Determining the Presence of the Venous Pulse.

I have already said that variations in the venous pressure can be recognised by changes in the venous pulse, and the questions naturally arise — Are coincident changes to be observed in the arterial pulse? and do the arterial pulses of patients with venous pulsation present a recognisable difference from the pulses of those in whom there is no such pulsation? Concerning this latter question no definite answer can be returned. The factors entering into the conditions that give rise to variations of blood pressure are so numerous and so indeterminate in their action, that no safe generalisation can be drawn from clinical observations. The individual pulse is such a distinct and definite entity, that no two persons possess exactly identical pulses, any more than they possess exactly similar noses. It is possible, however, that changes may be detected in individual pulses in the arterial pressure, coincident with those of the venous pressure, did we possess equally sensitive apparatus for registering them. Although I have spent a considerable amount of time in trying to ascertain this, I cannot yet speak confidently of success. This knowledge is to be obtained more from prolonged observations in individual cases, passing through a variety of conditions that will modify the circulation, than from cursory observations in a large number of separate individuals. So far I have attempted both, but the former of these two methods has yielded the more instructive results. It would be premature to give them, as they are incomplete, and their full import is not yet understood. I will therefore refer to a few points, in order to indicate rather what is the

[1] Coats, J., "Manual of Pathology," 2nd edition, London, 1889, p. 59.

nature of the changes and the factors producing them, than to make a futile attempt to settle the question definitely.

The cases in which the venous pulse occurs, apart from organic heart lesions, are those in which debility and cachexia are most marked features. Nevertheless, there are many patients who are debilitated and suffer from extreme cachexia who never present the phenomenon. I am unable to account for this difference, unless it be that the inherent cardiac tone is possessed of more enduring qualities. Bordering upon this class there is a large number of people, presumably in good health, who exhibit pulsation in the veins. Amongst these can be distinguished three classes in which this phenomenon is of very frequent occurrence, namely, fat people, chlorotics, and pregnant women. The type of the venous pulse in these cases, it is scarcely necessary to say, is invariably the auricular, and the character of the tracings obtained is always the same. In fat people the cause for its appearance is probably the loading of the heart by the increase of the pericardial fat, which embarrasses the heart's action. Under strict dietetic regimen, with resumption of greater freedom in respiration, I have noticed the venous pulse disappear. In chlorosis the general want of tone that is so evident is also present in the muscular tissues of the heart, leading to dilatation of the cavities, particularly the right, the area of the cardiac dulness assuming the form characteristic of increased size of the right side of the heart. As chlorosis is the disease to which so many observers have directed attention, as presenting symptoms of tricuspid regurgitation, the following case may be cited as an example of the form of venous pulse present in such cases. The venous pulse presents the characteristic auricular form, and it may be taken for granted that this form only occurs in chlorosis; the evidence of tricuspid regurgitation is to be sought for mainly in the ventricular wave.

Case 29.—Female, æt. 22; complains of shortness of breath and palpitation on exertion. The patient has always had good health, but during the last 18 months she has been getting weak and breathless. She is well nourished, and rather plump, but at times becomes very pale, flushing readily. The palpebral conjunctiva and mucous membrane of the lips are very pale.

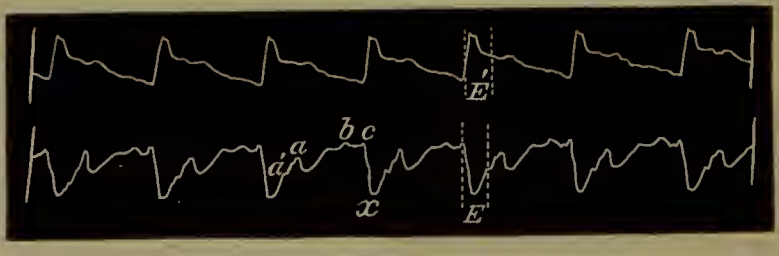

Fig. 90.—Tracings of radial and jugular pulses from a chlorotic female (Case 29).

The appetite is poor, and the patient suffers a good deal of epigastric pain after meals. The bowels are costive. The menstruation is regular but scant, and the discharge brownish. The pulse is rather small, soft, compressible, and regular, 72 per minute. There is well-marked pulsation in the veins of the

neck, which, upon a tracing being taken, is shown to be of the auricular type (Fig. 90). The heart impulse is distinctly felt, and the apex beat is situated in the fourth interspace, 3½ in. from the midsternal line. The heart's vertical dulness begins at the second interspace in the left parasternal line. It also extends about half an inch to the right of the middle line. The first sound is somewhat impure at the apex, and is replaced by a soft murmur at the base, heard best over the second and third left costal cartilages. There is a well-marked venous hum heard over the veins on the right side of the neck.

It is in pregnancy, however, and during the puerperium that the most instructive evidence of the venous pulse is to be found; the variations here are very striking, and are capable of being attributed to definite causes with less liability to fallacy. I have already referred to the fact that it was the study of the changes in the circulation during pregnancy that led me to pursue this inquiry into the venous pulse. It is generally recognised that changes do occur in the maternal circulatory system during pregnancy; and on *a priori* grounds, as well as on the occurrence of certain imperfectly understood—and usually misinterpreted—phenomena, it has been assumed that there occurs during this period a sort of physiological hypertrophy of the left ventricle. Undoubtedly a great number of changes do take place, but whether or not these point to the hypertrophy of the left ventricle does not concern the matter in hand. But what certainly does occur is dilatation of the cavities of the right heart. Although there are other concomitant phenomena indicative of this change, it would be unnecessarily introducing controversial matter, which would obscure the part played by the most striking of these, were the whole subject entered upon; and here only the venous pulse will be shortly dealt with. So far as I have been able to enter upon the study of this subject results have been obtained which, if properly understood, may throw much light upon the variations of arterial and venous pressure, and lead to a more philosophical appreciation of the forces at work in modifying the circulation. That the following interpretation is correct I am far from asserting, but it is such as occurs to me in applying what sum of knowledge I possess in this matter.

Occasionally women will pass through the whole pregnant and puerperal periods with no manifestation of the venous pulse whatever. Others will manifest it most strikingly during these periods, and for a long time after, while some will only manifest it at certain times. Usually those in whom this pulsation is wanting, or present to a slight degree only, are more vigorous than, and not so much troubled with breathlessness as, those in whom the phenomenon is more pronounced. This statement is not absolute, for numerous exceptions are met with. Still it may be stated as a general rule that those in whom the venous pulse is most developed present other symptoms indicative of cardiac weakness—breathlessness, œdema of the legs and lungs, and bronchitis. In such the venous pulse becomes most marked towards the end of pregnancy. The reason for this is to be found in the increased work thrown

upon the right heart on account of the diminished mobility of the lungs. From the pressure of the uterine tumour the respiratory excursions are limited in extent, are shallower, and are more rapid on exertion. The chest wall is opened up, and the chest circumference at its lowest part is widened. The elastic recoil of the cartilages is thus interfered with, and it is weeks or even months after delivery before the chest wall returns to its natural circumference.

Further, the spirometric observations of Dohrn[1] show that the vital capacity of the lungs is diminished before childbirth in the majority of cases. Of necessity the influence of the respiratory movements in the pulmonary circulation will be diminished, and more work thrown upon the right heart. The nutritive value of the blood in pregnancy appears also to be below the normal, and, in consequence, a deficient nutrition tends to induce weakness in the cardiac muscle, so that at the time more work is thrown upon the heart it is less able to bear the strain; it therefore dilates, permits tricuspid regurgitation, with the development of murmurs, and the venous pulse. It is in the later stages of pregnancy, too, that the early signs of pulmonary œdema, already referred to, are most instructively developed, and, in some cases, it is possible to demonstrate the concurrence of the venous pulse and the basal crepitations, both phenomena proceeding, *pari passu*, under the varying influences of the pregnant and puerperal states. Even admitting the possible element of the total increase in the amount of blood during the later stages of pregnancy, the above reasoning would still hold good, inasmuch as greater increase in bulk would not of itself determine the conditions necessary for the production of a pulsation in the veins, in which case it would be a constant phenomenon, which experience shows it is not. It is certainly best developed during the later stages in those anæmic women in whom the venous pulse was present, to a slight extent, before conception.

In a considerable number of patients in whom the venous pulse has thus developed during pregnancy, it disappears entirely during labour. It may persist during labour and disappear immediately after the child is born. The probable reasons for this disappearance will be discussed in the next section. On the other hand, it may reappear, or indeed appear for the first time during the puerperium, usually within 48 hours, but sometimes after 10 days. It is difficult to account for this satisfactorily, and the reason that seems most sufficient is that it is due to a weakness of the muscular walls, a sort of reaction after the excessive exertion undergone during labour. The consideration of other facts connected with the symptoms during this period seems to show that this view is reasonable. The patients seem weaker after the labour; sometimes they have attacks of syncope during the first few days of the puerperium, and frequently exhibit the symptoms of slight pulmonary œdema. Further, it is during the first 10 days of the puerperium that

[1] *Monatschrift für Geburtshülfe*, December 1886.

decided symptoms of heart failure are shown in those cases where the pregnancy has been complicated with valvular heart disease. Usually the venous pulse persists in a more or less marked form for a long period after the puerperium. My observations, carried out in numerous individual cases extending over a number of years, lead me to consider that the heart hardly ever regains its wonted vigour after a series of pregnancies, and that a frequent cause of respiratory troubles in multiparous women in later life is to be found in the cardiac changes during pregnancy. This seems to approach the conclusion arrived at by Allbutt, who, studying the subject from other aspects, says: "On the other hand, I have never seen evidence of tricuspid incompetence in hearts assuredly sound, although I have looked for it in scores of labourers and athletes under exertion. Whenever the venous reflux has been distinctly visible I have been able to recall it quickly in the same persons after long intervals of rest; showing, so far as this symptom has value, that the right auriculo-ventricular orifice is, in such persons, permanently weakened."

The recalling of the reflux noted here is well illustrated in Figs. 24 and 61 (Cases 15 and 17). I have also noticed the venous pulse produced in patients undergoing a prolonged and exhausting illness, as in typhoid fever when, as is well known, right heart dilatation occurs, and in convalescence from exhausting illnesses. But as yet my results are not sufficiently numerous to enable me to enter into this part of the subject further than to indicate the fact.

Section XVIII.—Conditions Determining the Disappearance of the Venous Pulse.

All circumstances tending to favour the strengthening of the heart's action assist in inducing a diminution of the venous pressure and a disappearance of the venous pulse. Hence, when it is due to organic heart disease, the treatment appropriate to the restoration of the destroyed balance of compensation tends to aid in reducing the venous pressure. In like manner, when it is present in cases where the heart is functionally weak, the measures suitable for the restoration of the patient's strength reduce the venous pressure, as in chlorosis, by the administration of iron and other appropriate remedies; in fat people, by the reduction of the fat by dietetic means; during convalescence from an exhausting illness, by the gradual resumption of a healthy action of the whole system, and coincidently of the heart's action. But besides this natural healthy mode of reduction of the venous pressure there are other circumstances under which the venous pulse may disappear. To me it was difficult to account for its disappearance at times when on *a priori* grounds I had thought it would have been more strikingly developed. The explanation, however, appeared clearer when I studied the manner in which the heart responded to certain stimuli, as illustrated more

particularly in the experimental researches of Roy and Adami. In referring to the subject of the influence of the various nervous mechanisms upon the circulatory system I am conscious of entering a region where numerous pitfalls are laid for the footsteps of the unwary. But there are safe grounds for drawing a limited number of conclusions. From the results of the observations of these two authors, it is shown that slowing of the heart induced by vagus stimulation increases the venous pressure and diminishes the arterial—the output of the heart being lessened as less blood enters. On the other hand, stimulation of the *nervi augmentores*,[1] although only slightly increasing the rate of the heart beat "increases the output of the heart by increasing the force of the auricular contraction, causing at the same time a rise of blood pressure in the pulmonary and systemic arteries, and a fall of pressure in the systemic and pulmonary veins." At the same time the ventricles dilate more and contract more forcibly and completely, the capacity of the heart and its capability for work being increased.[2] Coming to the clinical consideration of the subject it will be found that the circumstances that give rise to an increased activity of the heart tend to favour the disappearance of the venous pulse. The most striking of these is the increased action of the heart due to a rise in the body temperature. I have frequently had occasion to remark the complete disappearance of the venous pulse during a febrile attack. The reason for this is clear from the foregoing explanation. Not only is the capacity of the heart increased but also that of the arterial system. The large full pulse of the febrile condition implies increased capacity of the arteries. If, then, both the heart and the arterial system are more capacious, that alone is sufficient to account for an abstraction of the blood from the venous system with a disappearance of the venous pulse.

The following case illustrates the disappearance of the venous pulse, due to rapid action of the heart on account of the febrile condition :—

Case 30, æt 26, was safely delivered of a full-timed child (first). From the early stages of the labour there was no venous pulse present, nor was any trace observed till the 10th day after confinement—when a well-marked tracing was

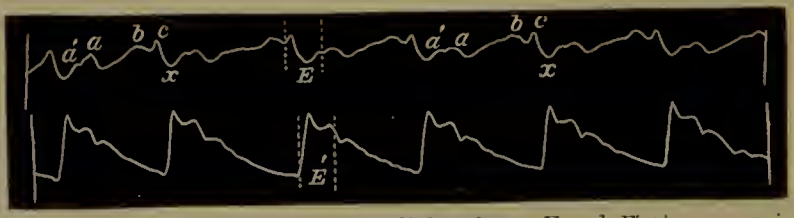

Fig. 91.—Tracings of the venous pulse and radial pulse. *E* and *E'*, time occupied by the ventricular outflow through the pulmonary and aortic orifices; *a'*, ventricular wave before, and *a*, after the closure of the pulmonary valves; *b*, auricular wave; *c*, arterial wave; *x*, auricular depression (Case 30).

obtained, similar in all respects to Fig. 91. The patient had prospered favourably, the temperature remaining normal, and no abnormality could be detected

[1] *Phil. Trans. Roy. Soc.* 1892, p. 243. [2] *Loc. cit.* p. 244.

in the heart or lungs. The pulse was usually somewhat irregular, the length of the beats varying as in Fig. 92. On the 16th day after confinement the

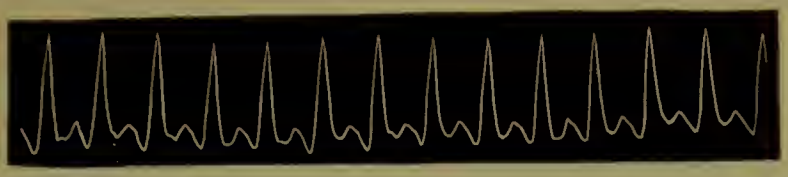

Fig. 92.—Sphygmogram from Case 30. Temp. 103° Fahr. ; pulse, 124 ; no venous pulse.

patient was seized with a severe rigor, the temperature rose to 103° Fahr., pulse 124 per minute, large, full, soft, and dicrotic (Fig. 92). There was not the faintest trace of a venous pulse. The following day the temperature fell to 99° Fahr., and there was a slight return of the venous pulse. On the second day after the rigor the temperature was normal, the pulse was 78 per minute, quiet and slightly irregular, much smaller in size but not so soft, and the venous pulse was well marked (Fig. 91).

In this case the degree of venous pressure must have been but slightly increased, and the heart capable of increased exertion. The reason for the disappearance of the pulse during such a febrile attack as the above and during exertion (as in labour) is evidently the same, and for the following reasons:—"The increase of the work performed by the heart is not absolutely proportionate to the increase in the force of the heart's contraction, the result of which is that sometimes the increase in force of the ventricle more than counterbalances the heightened resistance to contraction of the left ventricle, due to the increased contraction volume and rise in pressure in the systemic arteries, so that this part of the heart contracts more completely, having less residual blood than was the case before the work was increased.[1]" With this increased activity of the ventricle we have necessarily an increased arterial and diminished venous pressure, which is sufficient to account for the disappearance of the venous pulse during labour, as already noted.

While with fever and increased work the venous pulse may disappear, it does not always do so. If the heart's condition is so weakened that it does not respond to the extra stimulation with sufficient strength, the venous pulse may become distinctly exaggerated. This fact which I have also noted during labour and during a febrile attack (see the two following cases) confirm Roy and Adami in the further assertion.[2] "In other cases the increased force of contraction of the left ventricle does not fully counterbalance the increased work which is thrown upon it, and the ventricle does not contract so completely against the increased resistance, so that the amount of residual blood is increased. In other words, the increase in force due to the augmentors does not prevent the heart undergoing a certain degree of dilatation." It would be safe to infer that if the venous pulse be very well marked, and the venous pressure high, the changes due to fever or extra work of the heart are not sufficient to induce the disappearance of the venous pulse, but may

[1] Roy and Adami, *loc. cit.* 1892, p. 378. [2] *Ibid.* p. 272.

even exaggerate it. This is probably the explanation of the symptom observed by Lancisi, where in chlorotic females, on exertion, the venous pulsation was exaggerated (see Introduction).

In the following case fever and exertion failed to cause the disappearance of the venous pulse, although some other condition inducing rapid action of the heart succeeded in so doing :—

Case 31.—Male, æt. 18; examined 7th February 1892; complains of weakness and cough. This cough has persisted since an attack of measles in childhood. The patient is spare and thin. The pulse is soft, regular, and easily excited (as by coughing). There is a marked pulsation of the veins in the neck, giving a tracing similar to Fig. 93 when he is excited, and to Fig. 97 when the pulse is quiet.

Fig. 93.—Phlebogram of jugular pulse (auricular type) during excitement (Case 31).

The diffuse apex beat can be felt in the sixth interspace, and the heart's dulness extends 1 in. to the right and 3 in. to the left of the middle line. There is no murmur, and the first sound is very indistinct. Under suitable treatment the patient's general health materially improved until November 1892, when he had a mild attack of rheumatic fever. At the examination, made at this date, the temperature was 101°·5, and the pulse rate was 93 per minute, regular, soft, and slightly dicrotic. The venous pulse was well marked,

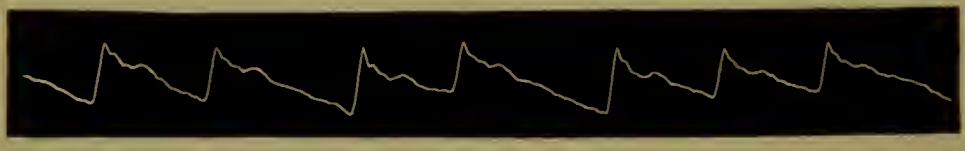

Fig. 94.—Sphygmogram of irregular pulse during convalescence from rheumatic fever (Case 31).

the tracing resembling in all respects that taken when he was excited and without fever (Fig. 93). The heart's condition was similar to that in the first examination—there being still no murmur. Under treatment by salicylate of soda the fever had nearly left him by the 30th, when the pulse was slow and irregular, as in Fig. 94. The temperature was 99.

The venous pulse was well marked all through the long illness. On the 3rd

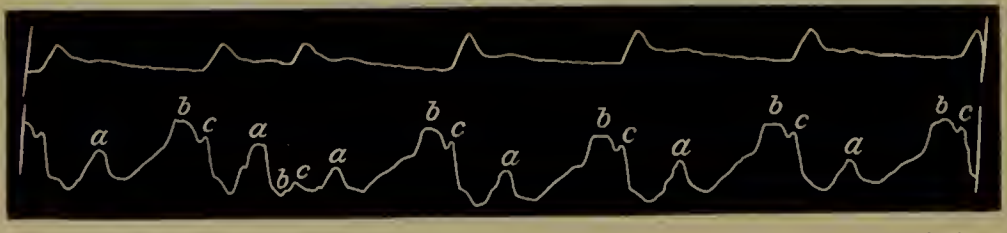

Fig. 95.—Tracings of radial and jugular pulses, showing correspondence in irregularity of both sides of the heart (Case 31).

of December the temperature was normal, the pulse still slow and occasionally irregular, 60 per minute. The venous pulse was still well marked (Fig. 95).

The radial sphygmogram here is not a good one. At this time I employed a sphygmograph with clockwork attached for marking time, which made the instrument very heavy and difficult to maintain in its place on a thin arm. Otherwise the features of the radial pulse resembled that in Fig. 94. The patient now made a rapid recovery, the arterial and venous pulses presenting the above characteristics. The heart never showed any symptoms of being affected. The salicylate of soda was stopped on the 25th of November. The patient was up and going about, and came to see me on the 17th of December feeling quite well. The radial pulse was, however, very soft and compressible, and beating at a great rate, with frequent irregularities (Fig. 96).

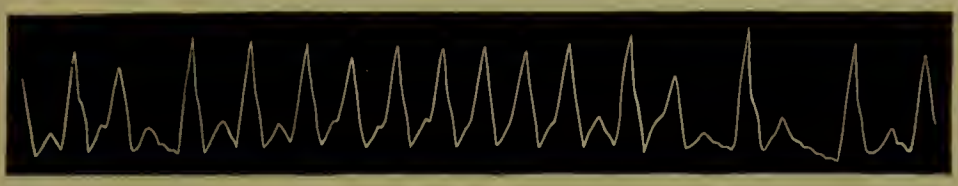

Fig. 96.—Radial sphygmogram during a period of rapid and irregular heart action, with disappearance of the venous pulse (Case 31).

There was now no venous pulse visible, and tracings of the pulsation in the neck showed no pulsation in the jugulars. This condition lasted for several days. On the 24th the pulse was soft, quick, and regular, but still no venous pulse was present. On the 27th the pulse was quieter and smaller, beating 80 per minute, and the venous pulse had returned. The patient has been under observation for some months, and the last time he was seen (5th May 1893) the tracing (Fig. 97) was taken, showing the usual features of the auricular venous

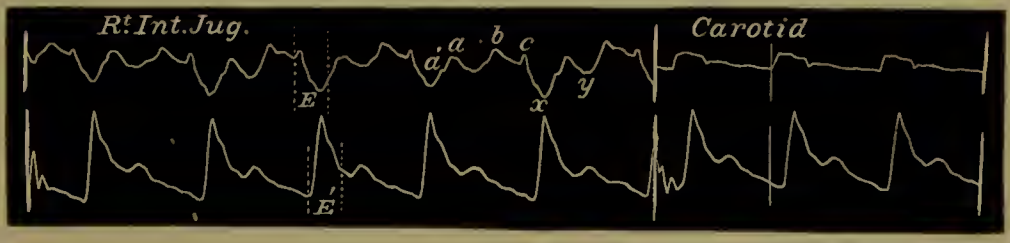

Fig. 97.—Simultaneous tracings of venous and radial pulses and of carotid and radial pulses after recovery from rheumatic fever and subsequent irregularity of the heart (Case 31).

pulse. No change had taken place in the heart's condition. The patient was looking in good health, and was following his work. There was a well-marked reduplication of the second sound.

(In so far as information about the difference in time of closure of the semilunar valves is to be derived from the study of the venous and arterial tracings no light is thrown upon the subject by the tracings. The interval of time between the reduplicated sound is so slight that it would be inappreciable on the tracing, even if one were absolutely certain of the exact period of the time of closure of the aortic valves.)

The points worthy of note in this case are — first, the exaggerated venous pulse on exertion and during the febrile stage of his complaint; secondly, the marked irregularity when the pulse became slower, with the venous pulse following the same irregularity, showing that the synchronism of the right and left hearts and of the co-ordinated movement of the auricles and ventricles persisted during this period of

irregularity; thirdly, the period of marked rapidity and irregularity, when the patient was free from fever and feeling comparatively well, and during that time disappearance of the venous pulse.

Concerning this last condition it would seem as if the capacity of the heart and the arterial system had so much increased as to reduce the excess in the venous system. The arterial tension here was not increased, for the pulse was so soft that a tracing could only be obtained if the lever was allowed to rest very lightly upon the artery, and to the touch the artery was large and full but very soft and compressible. Doubtless there was here great relaxation of the peripheral arteries and arterioles. According to the observations of Roy and Adami increased output of the heart does not necessitate a rise in arterial pressure, provided there is a relaxation of the vessels.[1]

Another instance of the disappearance of the venous pulse is seen immediately after delivery, when it has persisted during labour. The cause of this may be either the loss of blood or the sudden diminution of the intra-abdominal pressure. That the first of these two causes would be effective in reducing the blood pressure, venous or arterial, is self evident. But under the particular circumstances the removal of the abdominal tension would also greatly assist in diminishing the venous pressure by permitting greater distension of the capacious abdominal veins, as well as by freeing the chest from embarrassment. That this simple removal of an abdominal tension would suffice appears evident from the following case, where the removal of a large ovarian cyst was coincident with the disappearance of the venous pulse. This could not be attributed to the loss of blood, for the amount lost could not have exceeded more than a few ounces.

Case 32.—Female, æt. 44, married; examined 14th October 1892; complains of pain across the lower part of the abdomen and great swelling of the abdomen. She has had 7 children, the last being born 9 years ago. She has only menstruated 6 times since, but during the past 6 weeks she has lost a large quantity of blood. The swelling was first noticed last June, and has appeared to grow since then. The patient is pale and thin. The pulse is 120 per minute, soft, and regular. The veins of the neck are full and pulsate markedly, giving tracings of a characteristic auricular type. The heart presents no abnormality. The temperature is 102°·5 Fahr. The abdomen is greatly distended and tender to pressure. There is resonance in either flank, and the dulness in the middle line extends from above the umbilicus to the symphysis pubis. There is a sense of elastic resistance over this area, and the upper margin is felt definite and rounded off. For the next 14 days the patient's condition did not alter, but after that time the temperature fell. From this time to the 9th of November she gradually regained strength, the temperature kept normal, the pulse fell to 80 per minute, and the pulsation in the veins of the neck, though still evident, was less marked. On this date I operated, and removed a large multilocular ovarian cyst. The size of the tumour necessitated the prolongation of the incision to near the ensiform cartilage. The tumour was adherent in nearly its whole extent to the surrounding structures. Most of the adhesions readily gave way, but many were secured with catgut ligature.

[1] *Loc. cit.* p. 272.

The whole breadth of the omentum had to be systematically ligatured and cut. Notwithstanding this very little blood was lost. The patient made an uninterrupted recovery, but I noted the fact that from the time of the removal of the tumour there never was the slightest evidence of venous pulsation during the succeeding 2 months. A small enlargement of the right lobe of the thyroid pushed the carotid outwards, so that it lay along the posterior border of the sterno-mastoid muscle, and after the operation it became distinctly visible in the neck, whereas heretofore it had been obscured by the distended vein.

There are other circumstances that would appear to favour the disappearance of the venous pulse, but inasmuch as the causes producing them, and the conditions under which they occur are obscure, and I have only had isolated instances under observation, I do not deem it advisable to enter into any discussion regarding them. The whole subject is one of great difficulty, and in trying to reason it out one is continually liable to arrive at a fallacious conclusion through leaving some factor out of consideration.

Section XIX.—The Non-Rhythmic Contraction of the Cardiac Cavities.

It has long been a subject of much discussion whether it is possible for one half of the heart to contract while the other stands still. On the one hand, clinical observers have been found who have not hesitated to assert that they have seen, heard, and felt the two sides of the heart contract independently; on the other hand, it has been asserted as positively that such discordance in the heart's action is incompatible with life. A recent writer[1] on this subject summarises as follows:—"Only under abnormal circumstances, where there exists a considerable difference in pressure between the right and left ventricles, where the heart has been poisoned, or where it is dying, can it be possible for one ventricle to remain quiet while the other beats (hemisystole), or that a contraction limited to an individual portion of the heart chamber can take place." It is very doubtful if the evidence on which many clinical observations demonstrating the lack of harmony between the action of the ventricles are based has been trustworthy. Many of these observations have depended upon the study of the movements of the heart itself. But, unfortunately, interpretation of the cardiac movements is so surrounded with difficulties that little reliance can be placed on observations based upon the evidence afforded by the usual means of clinical examination. It seems late in the day to begin discussing the cause of the heart's impulse, nevertheless the subject is yet far from being properly understood, and until the various movements produced by the heart's action are understood, little or no reliance can be placed upon the results referable to this intricate subject, obtained by the usual method of examination. The interpretation of the quickly succeeding events in a cardiac revolution will baffle the most

[1] Von Frey, *loc. cit.* p. 73.

acute observers, unless, indeed, a preconceived idea of what is expected to happen be present in the mind. In such case, there is no lack of definiteness in regard to the results obtained. But a study of the views held upon the cardiac movements by writers of repute reveals a curious confusion in regard to these phenomena. It would seem that because the left ventricle in its contraction impinges against the chest wall, it must at the same time produce expanding impulses in every other direction. Thus, according to Walshe,[1] "During systole the ventricles *shorten and at the same time bulge* in all directions." Apart from the contradiction apparent in the italicised words in this sentence, it conveys the idea present with nearly all writers. And yet, if it be considered that the ventricular cavity is emptied during systole, the idea that it enlarges or bulges is quite out of the question. Because of the fact that the apex beats against the chest wall, it is tacitly assumed by most writers that, therefore, the left ventricle not only beats in every other direction, but that the other cavities may rise up and beat against the chest wall. If one were to say that a hollow viscus like the bladder, during the evacuation of its contents, "bulged in all directions" and beat against the abdominal wall, then all would look upon the statement as absurd. Yet, somehow, when dealing with the evacuation of the contents from the chambers of the heart, it appears to be necessary to assume the occurrence of some special unknown agency to enable the heart to act contrary to the laws of nature.

Balfour[2] asserts that the left auricle may be felt in certain cases beating against the chest wall immediately before the apex beat. The same author also elaborates the theory that a regurgitant mitral murmur may be heard in the second left interspace,[3] and adduces as evidence in support of this, the fact that the auricle has been found by Gibson[4] beating in the second left interspace at the same time as the apex beat on account of the regurgitation of blood into the auricle through the mitral orifice—a view previously expressed by Blakiston.[5] Valuable as Balfour's theories are, it can scarcely be expected that the auricle will be so accommodating as to beat the one time during its systole, and at the other during its diastole, in order to suit these theories. Like Balfour, Sansom[6] has also found the auricles beating before the ventricular systole, as low as the fourth rib. Yet in all the post-mortem records I have read, I have never heard of the auricles being found in this position. In one of Balfour's cases,[7] where this feature was present

[1] *Loc. cit.* p. 26.

[2] *Loc. cit.* p. 132.

[3] Balfour, G. W., "On the Position and Mechanism of Hæmic Murmur," *Lancet*, 15th Sept. 1877. Also *Edin. Med. Journ.* Sept. 1883.

[4] Gibson, G. A., "On the Rhythm of Auricular Impulses," *Edin. Med. Journ.* 1878, p. 1012.

[5] Blakiston, P., "Clinical Observations on Diseases of the Heart and Thoracic Aorta," London, 1865.

[6] Sansom, *loc. cit.* p. 126.

[7] Case 14.

to the last, no mention is made of the position of the left auricle in the post-mortem examination, which, considering the extraordinary position of the organ, would probably have been noted. Russell[1] has disputed these views of Balfour's and, recently, Harris[2] has thrown much doubt on the possibility of such an occurrence by careful observation on the post-mortem examination of suitable cases. I can support his results, for in such cases as 1, 3, 6, 18, and 46, particular attention was paid to the position of the cardiac cavities, and while the right auricular appendages sometimes encroached upon the left second interspace, in no instance did the left auricle approach the surface. Further, the study of the position of the organ in such excellent descriptions as that of Luschka[3] shows the improbability of such an occurrence. What I suspect these observers did find was the expansion of the ventricles during their diastole. Cruveilhier,[4] in a case of ectopic heart, noted that the diastole of the ventricles was rapid and energetic, so much so that at first sight it seemed to constitute the active movement of the heart, for the hand closed upon it was opened with violence. In like manner the diastole of the auricle occurred rapidly. Generally speaking, the study of the movements of the ectopic heart reveals the systolic shrinkage of the cavity. (Cruveilhier, Skoda,[5] Mitchell,[6] François-Franck,[7] Ziemssen,[8] and Gibson and Malet.[9]) In Ziemssen's tracing of the supposed auricular impulse, the systole is represented as a gradual rise, while in Gibson's curve[10] it shows a systolic depression, which also is the case in Porter's[11] tracing of the movements of the auricle, while Foster[12] describes the auricular appendages as being drawn in during their systole. Macdonnell's[13] tracings from the posterior and under surface of the heart are unsatisfactory, inasmuch as they are not timed with any

[1] Russell, W., "The Murmurs of Debility in the Pulmonary and Tricuspid Areas," *Edin. Med. Journ.* Aug. 1882 and Nov. 1883.

[2] Harris, T., "Some Clinical Post-Mortem Observations on the Cardiac Dulness in cases of Mitral Disease and Cardiac Dilatation, etc." *Medical Chronicle*, February 1893.

[3] Luschka, H., "Die Brustorgane des Menschen in ihre Lage," Tübingen, 1857.

[4] Cruveilhier, "Note sur les mouvements et sur les bruits du cœur," *Gazette Médicale de Paris*, 7 Août 1841.

[5] *Loc. cit.*

[6] Mitchell, T. R., "Remarkable Case of Arrest of Development in a Fœtus," *Dublin Quart. Journ. of Med. Sc.* 1st Nov. 1844, vol. xxvi. p. 262.

[7] "Nouvelles recherches sur un cas d'ectopie cardiaque (Ecto-cardie)," *Archives de physiologie normale et pathologique*, 1889, p. 70.

[8] Ziemssen, v. "Studien ueber die Bewegungsvorgänge am menschlichen Herzen, sowie ueber die mechanische und elektrische Errigbarkeit des Herzens und des Nervus Phrenicus, angestellt an dem freiliegenden Herzen der Caterina Serafin," *Deutsches Archiv f. klin. Med.* 1881, Bd. xxx. s. 270.

[9] Gibson, G. A., and Malet, "Presternal Fissure uncovering the Base of the Heart," *Journ. of Anat. and Phys.* Oct. 1879, vol. xiv. p. 1.

[10] Gibson, G. A., *Edin. Med. Journ.* 1878, p. 1012.

[11] Porter, *loc. cit.*

[12] Foster, M., "A Text-Book of Physiology," 6th edition, 1893, p. 232.

[13] Macdonnell, H., "Cardiograms from the Human Heart," *Practitioner*, 1890, vol. xliv. p. 178.

event to show whether the rise in the tracings is really systolic, as he assumes it to be. Romberg holds to the view based on the study of the disposition of the muscular fibres that the long axis of the left ventricle remains the same during systole and diastole, while the other two diameters diminish during systole. The right ventricle shortens, and the *conus arteriosus* at the same time becomes flatter during the systole. It is difficult to arrive at a distinct idea of what really takes place, and of the causes of the combinations of impulses that result from a cardiac revolution, but it seems safe to assume that if from the inherent structure of any chamber it is made to move in one direction, it must recede from the opposite, and that its diameter must diminish with the evacuation of its contents, and increase with its filling. There is no proof that I have been able to find which shows that any chamber but the left ventricle is able to give a distinct impulse to the chest wall during its systole, and the disposition of its muscular fibres is the undoubted cause for this. I have already dealt with the fact that the ventricular diastole does undoubtedly produce an impulse sufficient to cause an appreciable descent of the liver into the abdominal cavity.

The reason I digress somewhat from matters germane to my subject is, that hitherto the impulse of the heart against the chest wall during ventricular systole has been assumed to be due in some cases to the contraction of the right ventricle alone. Thus Leyden[1] has taken tracings of contractions of the right ventricle when the left was supposed to be in abeyance, and other writers describe a pause in the radial pulse, while a weak impulse was felt over the chest,—supposed to be indicative of a contraction of the right side of the heart, while the left stood still. Additional evidence for this has been found in the fact that during this pause of the left heart there has sometimes been noted a persistence of the venous pulse. I will endeavour to show that this is no evidence of a cessation of the left heart's action, but that the movement felt in the chest at that time may have been due to a feeble contraction of the left ventricle. In the following case it was distinctly noted during a pause in the radial pulse that an impulse, much weaker than usual, was communicated over the front of the chest. A tracing taken of the apex beat shows that during the pause in the radial pulse, it was distinct but small. In later observations when (under the influence of digitalis) the heart's action was stronger, these pauses were interrupted by small pulse beats, giving rise to the form of pulse known as the *pulsus bigeminus.*

Case 33.—Female, æt. 34; examined 2nd July 1892; complains of great weakness and shortness of breath and of palpitation on the slightest exertion.

[1] Leyden, E., "Ungleichzeitige Contraction beider Ventrikel, *Virchow's Archiv,* 1868, Bd. xliv. s. 365; and "Zwei neue Fälle von ungleichzeitige Contraction beider Herzkammern," *Virchow's Archiv,* 1875, Bd. lxv. s. 153.

The patient has had 5 children. The fourth child was born 7 years ago. She has been a strong healthy woman up to 2 years ago, when she became pregnant, and during the whole of the pregnancy she was very sick, and very short of breath; towards the end she swelled very much in the feet and legs. She had a rapid and easy labour, and felt better for the first week, but after that she kept getting worse, swelling of the legs and shortness of breath coming on. In February last she had a stroke; losing the power over the left side of the body, and was laid up for 6 weeks. She still remained very weak. The patient is of medium height, rather stout; lies in bed, requiring to be propped up, and is seized with great dyspnœa and palpitation on the slightest exertion. The swelling has mostly disappeared from the legs. The pulse is small and very irregular in the sphygmogram, showing little runs imperceptible to the finger. There is no fulness of, and no pulsation in, the veins. The chest is animated by irregular series of shocks from the heart's contraction, and only a very indefinite apex beat can be felt in the sixth interspace. The area of the heart's dulness begins vertically in the second interspace in the left parasternal line, and extends from half an inch to the right of the middle line outward to the left for 6 in. The sounds are free from murmurs, but it is difficult to identify the individual sounds.

The progress of the case was very slow, and during the time numerous careful observations were made. The pulse was always very irregular, and sometimes a definite apex beat would be present, from which fair cardiograms could be obtained. Towards the end of the month she recovered considerably, when there could be distinguished distinctly two murmurs at the apex, and during a few regular beats these could be recognised as being presystolic and systolic in rhythm. She again came under observation on 10th March 1893, when the cardiac condition was found very much as described above. In feeling the pulse, however, distinct intermissions could be detected, and in applying the left hand over the præcordia at the same time as the right index felt the pulse, a slight shock was communicated to the hand at the time of the intermission in the radial pulse. Several tracings of apex beat and radial pulse were taken by the clinical polygraph, all presenting the same features as that in Fig. 98. By pushing the finger into the upper part of the epigastrium and to the left, the heart impulse was distinctly felt. A tracing taken of the impulse here, by means of a Knoll's cardiograph, revealed features identical in time and character with those present in the apex-beat tracings taken at the same time. No liver pulse could be felt. The patient was prescribed a mixture containing digitalis, and she gradually improved, and tracings of apex beat and radial pulse always showed synchronous but irregular action.

FIG. 98.—Simultaneous tracings of the apex beat and radial pulse, showing small ventricular contractions during pauses in the radial pulse (Case 33.)

I have not sufficient evidence by me to assert dogmatically in what manner the heart chambers, other than the left ventricle, would exhibit their movements

on the chest wall. That distension of the right heart does communicate movements I have no doubt. I have found a diastolic epigastric impulse due to enlarged right ventricle. I have also, many times, felt the heart movement to the right of the sternum appear with dilatation of the right heart and disappear with improvement in the heart's condition, but I was never able to time it to my satisfaction, and the

FIG. 99.—Simultaneous tracings of pulsation in the right internal jugular vein (upper tracing) and of the apex beat (lower tracing), showing a persistence in the venous pulse during a pause in the ventricular systole (Case 45).

doubt was present in my mind whether it was not a communicated movement from the left ventricle. This doubt was strengthened from the following observation. In Fig. 99 tracings are given of the apex beat at the same time as the venous pulse—the latter being of the ventricular type (Case 45).

It will be observed that there are two beats of the apex followed by a pause. On the other hand, the venous pulse occurs in groups of three waves, showing two pulsations synchronous with the apex beat, and another pulsation (wave 3) during the pause in the apex beat. The descent of this latter wave is interrupted by the next wave, which takes on the same rhythm for the succeeding two beats. So far as one is able to interpret this tracing from what has gone before, it would be said that there is no doubt that the right ventricle is contracting here while the left stands still. The apex beat at this time was a very well-marked feature, and there was not the slightest difficulty in getting a tracing. Moreover, the right heart tends to regularity in action, inasmuch as the three waves of each group of the venous pulse occur at regular intervals. The succeeding wave occurs earlier to take on the rhythm of the left ventricle, which is stimulated to contract earlier than the normal rhythm (assuming one revolution to have been missed), on account, it may be, of the extra stimulus of the increased amount of blood. Such indeed is the explanation that appears most feasible, a distinct evidence of contraction of the right heart while the left stands still. But it so happened that a distinct impulse could be felt over and immediately to the left of the sternum, in the third interspace. I had no difficulty in getting a tracing of the underlying movement. Considering that the apex beat was well outside the nipple line, it

leaves little room for doubt that the right ventricle was situated immediately under the point where the tracings were taken. In the post-mortem examination it was specially noted that the right ventricle occupied the whole of the anterior surface of the heart to the left of the sternum. Inasmuch as the apex beat had disappeared before death, the right ven-

FIG. 100.—Simultaneous tracings of pulsation of the apex beat (upper tracing) and from the third left interspace near the sternum (lower tracing), showing simultaneous irregularity of both movements (Case 45).

tricle, no doubt, had increased in extent subsequent to this observation. Assuming then, as it is quite justifiable to do, that the tracing was obtained from the right ventricle it is found that during the pause at the apex there is a similar pause of the pulsation over the right ventricle (Fig. 100).

Numerous tracings were taken to verify this. If we assume, then, that the venous pulse was due to the right ventricle, the tracing in Fig. 100 demonstrates that the movement of the right ventricle was simply a communicated movement due to the more powerful left ventricle. No evidence is given of the right heart movement during this pause. These tracings do not demonstrate the direct effects of the movements of the right ventricle, but they do show that the impulse over the right ventricle may not be due to the contraction of this chamber. I do not wish to read more into these tracings than can be easily and logically demonstrated, but the occurrence of such changes is sufficient to give pause to the too hasty assumption, based upon the physical signs of the heart's impulse, that there is independent action of the heart chambers.

The possibility of the right and left hearts acting independently has also been assumed from the lack of harmony between the venous pulse and heart beat or arterial pulse. There is no doubt that the study of the venous pulse affords evidence far more trustworthy than the evidences of the obscure phenomena of the cardiac impulses. But in the records of cases where the discordance between venous and arterial pulses has been insisted upon as evidence either for or against the view of a rhythmic contraction of the two sides of the heart, two decisive points have generally been wanting, namely, the simultaneous record of both venous and arterial pulses, and the correct interpretation of the nature and form of the venous pulse. Sometimes the independent action of

the two sides of the heart has been diagnosed by observing the persistence of the venous pulse with absence of the radial. Frequently a tracing of an apex beat or arterial pulse has been taken and compared with the venous pulse taken at a different time, and the relation of the two sides of the heart interpreted by comparison of the tracings.

While both of these methods are valuable and instructive, as far as they go, there is so much left indeterminate that no safe conclusion can be drawn. The cases I shall give illustrative of the subject, with the exception of one case, will show either heart or arterial movements taken synchronously with the venous pulse, and the results will demonstrate a variety of variation in the actions of the cardiac chambers hitherto unsuspected. Indeed, so varied are these that, although my observations are more extensive than those of the majority of observers, I refrain from taking part in the controversy, and merely place them on record with such interpretation as seems to me most natural, but at the same time giving as accurate data as possible, so that others may verify my explanation, or find in the tracings material capable of other interpretations.

The discussion has centred mainly around that action of the heart which produces the form of arterial pulse, known as the "pulsus bigeminus." Although it is difficult to give a proper definition of this pulse, yet it is possessed of distinct characteristics. It represents the early occurrence of an imperfect systole following a normal systolic contraction of the left ventricle. It may occur at intervals of varying frequency in an otherwise regular pulse. It may be associated with other forms of irregularity, or the pulse may only consist of these two beats linked together. Frequently the smaller beat is not perceptible to the finger, but is usually demonstrable in the sphygmographic tracings. In the radial sphygmogram it presents a very characteristic appearance. The descending line of a normal pulse is interrupted after the dicrotic wave by an abrupt rise. I lean to this view of the pulsus bigeminus, namely, the abrupt rise in the descending line, this descending line continuing at nearly the same angle as it did before the interruption. But this description is not absolute, inasmuch as this abrupt wave in the descending line approaches and passes into a normal wave produced by an early occurring perfect systolic contraction. It is thus difficult to limit the definition; but I am disposed to consider the typical pulsus bigeminus as somewhat distinct from an unusually early occurring pulse beat, inasmuch as the latter presents all the features of the usual beat, namely, tidal wave, dierotic notch, and dicrotic wave; whereas in the bigeminal form the second wave is often a mere triangular elevation in the descending line of the first wave. In other words, the early occurring pulse beat represents a complete systolic contraction; whereas the small wave of the bigeminal pulse represents an imperfect systolic contraction. Of course in this there is still no distinct line drawn. Nevertheless, the fact remains that a

characteristic form is given to this pulse which permits of its easy recognition. Numerous definitions have been given of this pulse, but they are usually incomplete, leaving unexpressed some peculiarity or limitation. Perhaps Riegel, who more than any other has studied this subject, has given the fullest definition. "The pulsus bigeminus is characterised by the fact that after a strong systole of the heart, an incomplete diastole follows. The second incomplete systole occurs earlier, and is followed by a long and complete diastole of the heart."

Traube,[1] in his original description of this form of pulse, also included the "pulsus alternans"—the regular variation of a high and low pulse wave—as a form of pulsus bigeminus; but several observers incline to view it as quite distinct (Dehio,[2] Leuderitz,[3] Sansom[4]).

So far, my experience does not enable me to express an opinion. It is unnecessary here to enter fully into the mode of production of the "pulsus bigeminus" further than to state that in experimental investigations it is found to arise in numerous ways, as by the exhibition of drugs (digitalis[5] helleborine[6]), stimulation directly of the heart,[7] or of the vagus, injury to the heart, mechanical increase of resistance to its work,[8] etc. Concerning clinical observations, it is changed frequently into other forms of irregularity. When it occurs at intervals it may disappear simply with restoration of the heart's strength. Even in the purest form, when for weeks or months it may exhibit no variation, if watched long enough it ultimately becomes associated with other forms of irregularity (Case 38). In this my observations support those of Riegel. It has had attributed to it associations of the most varying qualities, being considered by its discoverer, Traube, as being of lethal significance; whilst Tripier[9] considered it, when not due to digitalis or valvular disease, to be associated with epilepsy. For a time it was considered significant of mitral stenosis. Numerous cases have been placed on record under different headings (allorhythmia, linked beats, coupled beats), all tending to show that no limited significance can be attributed

[1] Traube, L., "Ein Fall von Pulsus bigeminus nebst Bemerkunge ueber die Leberschwellungen, etc.," *Berlin. klin. Woch.* 1872, No. 16, p. 185.

[2] Dehio, K., "Ein fühlbaren Puls auf zwei Herzcontractionen," *Deutches Arch. f. klin. Med.* 1891, Bd. xlvii. s. 307.

[3] Leuderitz, C., "Ueber den Ablauf des Blutdrucks in Aortenstenose," *Zeits. f. klin. Med.* Bd. xx. Heft. 4–6.

[4] Sansom, *loc. cit.* p. 468.

[5] Boehm, R., "Untersuchung ueber die physiologische Wirkung der Digitalis und Digitalin," *Arch. f. d. ges. Physiol.* Bonn, 1871, Bd. v. s. 153.

[6] Knoll, P., "Ueber Incongruenz in der Thatigkeit der beiden Herzhälften," *aus den Sitzungsberichten d. kais. Akademie d. Wissenschaften in Wien, Mathem.-Naturw. Classe*, Bd. xcix. Abth iii. Januar 1890, Tafel iv.

[7] Marey, E. J., "La Circulation du Sang," Paris, 1881, p. 42.

[8] Rolleston, H. D., "Observations on Endocardial Pressure Curves," *Journ. of Anat. and Phys.* 1887, vol. viii. p. 235 (also, Leuderitz, *ante*).

[9] Tripier, R., "Des déviations du rhythme cardiaque, associées a l'epilepsie à la syncope. Phénomènes concomitants relatifs au pouls artériel et veineux, 1883, vol. iii. p. 1001 *et seq.*

to it. Still one gets into the habit of classifying symptoms empirically, when no true and fitting explanation is at hand, and I have myself for a long time regarded it as evidence of a jaded heart, for the reason that it appears in otherwise healthy hearts when submitted to a long strain, or to influence detrimental to its work (as chlorosis, fatty infiltration). It likewise makes its appearance in hypertrophied hearts, when compensation begins to fail. In looking over my notes and tracings of over 100 cases in which this form of pulse was present, I find it always associated with evidence of failure of heart power. Thus it occurs in heart failure from valvular disease, from compensation giving way in hypertrophy secondary to Bright's disease, atheromatous arteries, and indeterminate causes. Its most instructive appearance is in pregnancy, and after an exhausting illness (convalescence from erysipelas, influenza, bronchitis).

In regard to pregnancy its time of most frequent occurrence is during or immediately after labour. It may occur in the later months of pregnancy or during the puerperium, and in the latter case it may last for a considerable period, disappearing and reappearing at intervals. On the other hand, I have never found it in the irregular pulse of youth. Before puberty the pulse is very frequently irregular at times, independent of disease, and in perfect consonance with health. But although it may vary in rhythm, slow and quick beats alternating, I have never obtained a tracing showing the pulsus bigeminus unless there was valvular disease or dilatation from some cause. In like manner I have not found it in those attacks of irregularity dependent on gastric or other forms of reflex irritation. Futhermore, with rest and other treatment suited to the restoration of heart force, the pulsus bigeminus may disappear. It is mainly on these grounds that I have adopted for myself the provisional and purely clinical interpretation of the pulsus bigeminus as being the evidence of a jaded heart. This view is supported by the fact of its production experimentally by increased work thrown on the heart (as in obstruction of the aorta) and when the heart is dying.

In one of Hürthle's[1] tracings a very instructive illustration is given in regard to the method of production of the pulsus bigeminus. These tracings represent the ventricular and aortic pressures. After a normal contraction of the ventricle there are two smaller contractions. The first of these is just of sufficient strength to open the aortic valves, and in the aortic tracing there is represented a slight elevation, giving to the tracing the appearance of the pulsus bigeminus. The second of the imperfect ventricular systoles is not of sufficient strength to open the aortic valves, consequently no evidence of its occurrence is visible in the aortic tracing. Had it been a little more powerful the aortic tracing would then have presented the form characteristic of the pulsus trigeminus.[2] There is no doubt that this lack of power of the ventri-

[1] *Loc. cit.* Fig. 10, Tafel ii.

[2] See also Marey, *loc. cit.* Fig. 54.

cular systole to overcome the pressure in the aorta is the cause of many of the cases of hemisystole on record, where a pause in the arterial pulse has been accompanied by a movement of the heart or of the veins. On the other hand such a pause may ultimately be replaced by a slight elevation, as if the ventricular systole had increased in strength so as to be able to overcome the aortic pressure. (Case 33, see also Frey.[1]) Figs. 101 and 102 show probably the weakening of the ventricular contraction which is unable to open the aortic valves, for at such times the right ventricular action is present in the veins.

Sometimes in place of one beat on the descending line of a large arterial pulse there may be two or more, giving rise to the form of pulse known as the "pulsus trigeminus," "quadrigeminus," etc. Here, doubtless, as shown in Hürthle's tracing, already referred to, there are two or more imperfect systoles of the ventricle, and some of these not being powerful enough to open the aortic valves, the pulse becomes changed into a bigeminal or single pulse wave. This is well seen in Fig. 101, where the radial pulse is taken synchronously with the pulsation in the internal jugular vein. The tracing of the venous pulse is not very distinct, the circumstances of the case preventing the possibility of obtaining a better, still it will be noted that there is a distinct rise to every beat of the radial pulse in the earlier part of the tracing, considerably marred, it is true, by the respiratory movements. When the breathing is stopped the trigeminal pulse assumes first the bigeminal form, then, after a few irregularities, none of the lesser imperfect ventricular systoles appear, while all the time the venous pulse continues as before.

I do not wish to read more into this somewhat imperfect tracing than really occurs. I regret indeed having to use it, but it illustrates, notwithstanding its imperfections, the fact so apparent to the observer, that the veins pulsated distinctly during pauses in the radial pulse.

FIG. 101.—Simultaneous tracings of the radial and jugular pulses. The latter is of the ventricular type, and its character during the first part is obscured by the respiratory movements. The radial pulse, until the breathing is stopped, is of the trigeminal form. After the breathing is stopped the form of the irregularity of the radial pulse alters, and there are pauses, during which probably the imperfect ventricular systole is unable to open the aortic valves, for the venous pulse (and therefore the systole of the right ventricle) persists. (Reduced one-half.)

[1] Frey, A., "Pulsus rarissimus," *Berlin. klin. Woch.* 1887, No. 5, p. 72.

The veins were distinct in this patient, but so soft was the pulsation that considerable difficulty was at times experienced in getting tracings.

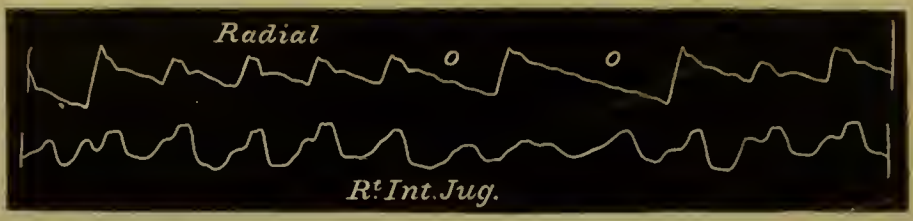

FIG. 102.—Tracings taken simultaneously of the radial pulse (upper tracing) and of the pulse in the right internal jugular vein (lower tracing). The venous pulse is of the ventricular type and continues during the pause (*o*, *o*) of the radial pulse (Case 18).

The venous pulse is of the ventricular form, although the characteristic features are not always readily recognised. The respiratory movements cause the large depression in the earlier part of the tracing, and to a certain extent confuse the reading. In some respects Fig. 102 shows the character of the venous pulse better, and here also in two places (*o*, *o*) there appear venous pulsations when there is a pause in the radial pulse. But not only did the radial and venous pulses show this difference, but latterly there was developed in this patient (Case 18) an utter want of synchronism between the two sides of the heart, as manifested by the carotid and jugular pulses. It will be noted in the earlier tracings from this patient, that there was perfect harmony between the arterial and venous pulses when both were present (Figs. 64–66). On two separate occasions there was present the utter discordance evident in Figs. 103 and 104. In these it will be observed that not only does the venous pulse persist during a pause in the carotid, but that they bear no relation whatever to one another in the time of their occurrence.

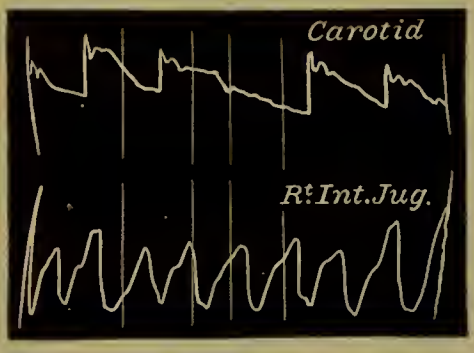

FIG. 103. — Simultaneous tracings of carotid and jugular pulsations, showing complete absence of harmony in their rhythm (Case 18).

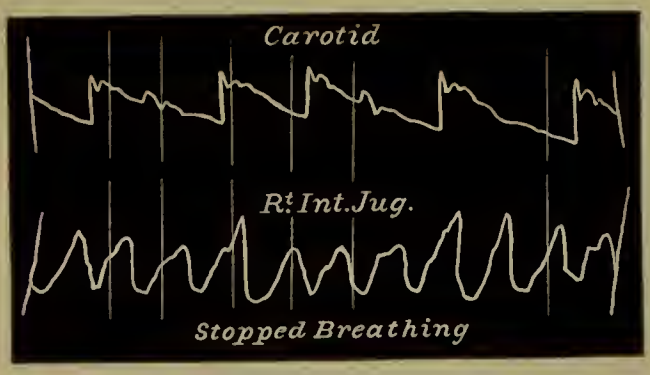

FIG. 104.—Showing the same features as Fig. 103 (Case 18).

There are three views in regard to the changes occurring in the heart during periods of irregular rhythm, based mainly on the evidence

of individual cases. First, that the two sides of the heart are beating at different times (Leyden,[1] Roy,[2] etc.); second, that there is an alternate systole of the two sides (Unverricht[3]); and, third, that the heart acts in the manner that produces the pulsus bigeminus, and the systole is at times so imperfect as not to have sufficient force to raise the aortic valves, but that the right ventricle is able to drive blood back into the veins. This last view is supported by the recital of many facts by Riegel[4] in his interesting monograph on this subject. It must be confessed, however, that strong as his arguments are his own tracings are far from conclusive, inasmuch as, first, neither the heart nor arterial pulse is taken synchronously with the venous pulse; second, no attempt is made to differentiate the individual characters of the venous pulse in order to estimate what may be auricular and what ventricular, and finally the variations that can and do occur are more numerous than he supposes.

The results I have obtained present such a variety of feature that it would be impossible for me to dogmatise in favour of any of the three views. Numerous forms of variation appear capable of production. But the great majority of writers have left out of consideration the part played by the auricle in the production of the symptoms observed, although it is the effect of the auricle's contraction that is in many cases the salient feature in the venous pulse. Before entering upon the account of my own observations of the pulsus bigeminus I may remark on the changes occurring in simple irregularities of the pulse. In cases where there is simply a lengthening or a shortening of the ventricular diastole, the right auricle and ventricle faithfully adopt the same rhythm. This is clearly evident in Fig. 79, Case 22, and Fig. 95, Case 31. Even when the pulse has been of the bigeminal type, agreement in rhythm is followed when an irregularity other than the bigeminal is present.

Case 34.—Female, æt. 30. I have attended this patient at intervals for nearly 10 years. On February 20th, 1891, I found for the first time an

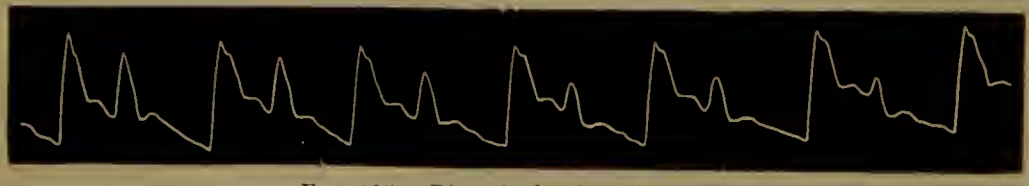

Fig. 105.—Bigeminal pulse (Case 34).

irregularity in the pulse; sphygmographic tracings showed the occasional presence of the pulsus bigeminus. The patient at this time was 8 months

[1] Leyden, E., "Ungleichzeitige Contraction beider Ventrikel," *Virchow's Archiv*, 1868, Bd. xliv. s. 365; and "Zwei neue Falle von ungleichzeitige contraction beider Herzkammern," *Virchow's Archiv*, Bd. lxv. s. 253.

[2] Roy, C. S., "On two Heart Cases which presented a rare Form of Irregularity," *Edin. Med. Journ.* Jan. 1878, vol. xviii. p. 594.

[3] Unverricht, "Ueber abwechselnde Zusammenziehung der beiden Herzhälften-Systolia alternans," *Berlin. klin. Woch.* 1890, No. 26.

[4] Riegel, Franz, "Zur Lehre von der Herzirregularität und Incongruenz in der Thätigkeit der beiden Herzhälften," Wiesbaden, 1891.

pregnant with her seventh child. The child was born on the 29th of March. On the fourth day after delivery the pulse was almost always of the bigeminal form (Fig. 105).

On no other date was there such a well-marked tracing obtained, but frequent subsequent observations revealed the occasional presence of this form of pulse. She again conceived, and was delivered on June 1892 (I did not attend the confinement, but was informed by the doctor in attendance that the pulse never presented the slightest irregularity). She again consulted me, January 21st, 1893, feeling weakly, and I found the occasional persistence of the pulsus bigeminus. There was present a faint venous pulse, and it was not easy to get a good tracing, but that in Fig. 106 shows clearly enough the relation of

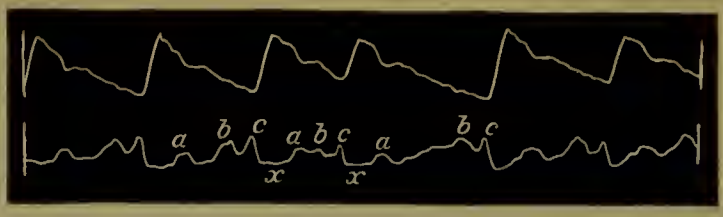

FIG. 106.—Simultaneous tracings of radial and jugular pulses. The venous pulse is of the auricular type, and during the period of irregularity accurately corresponds with the radial pulse in the time of its movements (Case 34).

events. The venous pulse corresponds with that of the radial. In the short beat the auricular wave *b* can be observed preceding the arterial wave *c*, the wave *a* preserving the same relative distance from *c* as the dicrotic notch of the small radial pulse does from the beginning of its upstroke. That is to say, the wave *a* representing the time of the closure of the pulmonary valves corresponds exactly with the time of closure of the aortic valves in the small beat as in the larger beats. No abnormality of the heart was ever detected in the patient, and the fact that she went through her successive pregnancies and attended to her numerous household duties (for she has worked very hard) testifies to her good health, though I have no doubt she is being worn down by these pregnancies and her hard work; of that this appearance of the pulsus bigeminus is most striking evidence.

During the period of the occurrence of the bigeminal pulse the vein may follow exactly the same time in its variations as in the following case:—

CASE 35.—Female, æt. 26; complains of huskiness and slight sore throat, which has troubled her for a year. She has had good health on the whole, and has never had any severe illness. For the past 2 or 3 years she has been troubled with shortness of breath and palpitation on exertion. The patient is very pale but fairly well nourished. The back of the fauces has a dry varnished appearance. The pulse is regular, but every second beat is a very small one, and occasionally not perceptible to the finger, presenting in the sphygmographic tracing the characteristic bigeminal form; there is no pulsation in the veins of the neck, but immediately above the clavicles marked pulsation, limited to the jugular bulb, is observed. To each beat of the pulse there is evidently a corresponding movement in the bulb, and the two beats in the bulb vary in size, a large one and a small. But the small one bears a distinct relation in time to the large radial pulse, while the large venous pulse is related to the small arterial pulse. This is very evident also in the tracing where the auricular wave *b'* is always highest immediately before the arterial wave *c'* corresponding to the lesser arterial wave in the radial pulse. It is to be noted also that there is a total absence of the ventricular wave *a* after the

large auricular wave. The heart's impulse presented the same form of irregularity. There was distinct enlargement of the area of the heart's dulness, the

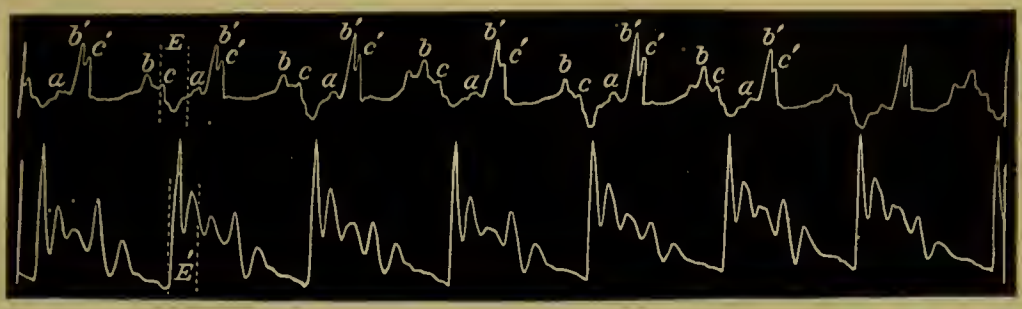

Fig. 107.—Simultaneous tracings of pulsations in the right jugular bulb and radial artery. The radial pulse is of the bigeminal form. *b′* and *c′* are the auricular and arterial waves, corresponding to the lesser radial pulsations, and it is to be noted that the auricular wave *b′* is much larger than the auricular wave *b*, preceding the larger radial pulse, and that after the waves *b′* and *c′* there is no ventricular wave *a*. On the ventricular wave *a* the time of closure of the pulmonary valves is well marked by the notch (Case 35).

vertical dulness in the parasternal line beginning at the second rib, the transverse extended from 1 in. to the right of the sternum outward to the left

Fig. 108.—Simultaneous tracings of pulsations in the right jugular bulb and in the radial artery, and of the carotid and radial. The slight movement of the venous pulse (to the sight representing a mere vibratory trembling) show all the features of the usual form of the venous pulse, and if carefully timed these movements can be readily recognised, even to the pulmonary notch in the ventricular wave *a* (Case 35).

for $4\frac{1}{2}$ in. There was no murmur, and the sounds could be heard quite distinctly, the first and second sound appearing normally, but quickly followed

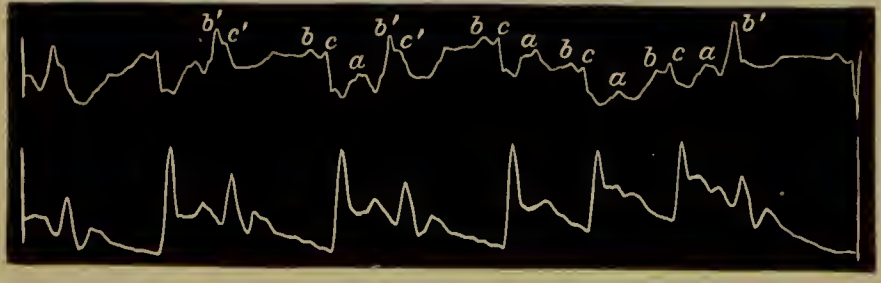

Fig. 109.—Simultaneous tracings of pulsations in the right jugular bulb and radial artery. The radial pulse passes into a more regular form for two beats, and the venous pulse likewise alters. It is to be noted that then the ventricular wave *a* appears, having been absent with the lesser beats, and the auricular wave *b* becomes smaller (Case 35).

by two short sharp sounds corresponding to the smaller pulse beat. On subsequent examinations this form of irregularity was not always present, the

radial pulse at times being quite regular, and the pulse in the jugular bulb then being less well marked, presenting in fact a mere tremulous vibration, but in the tracing each movement is capable of interpretation (Fig. 108). At other times the radial pulse presented transitional forms, the smaller beat increasing in size, sometimes presenting all the features of a normal pulse beat. At such times the auricular wave never rose so high as at the time of the small radial pulse of the bigeminal form—the ventricular wave being present (Fig. 109).

Case 36.—Male, æt. 51. Complains of shortness of breath and dropsy. The patient enjoyed good health up to this present illness, but he has been accustomed to free indulgence in alcohol. Four months ago he began to swell in the legs and face, and was laid up for a couple of months. He was able to move about freely till a week before the present examination. He is a stout man with a ruddy complexion. The face is now swollen, puffy, and of a livid red colour. There is a good deal of swelling under the left eye and of the left arm and both legs. The external jugular veins are full and distended, and

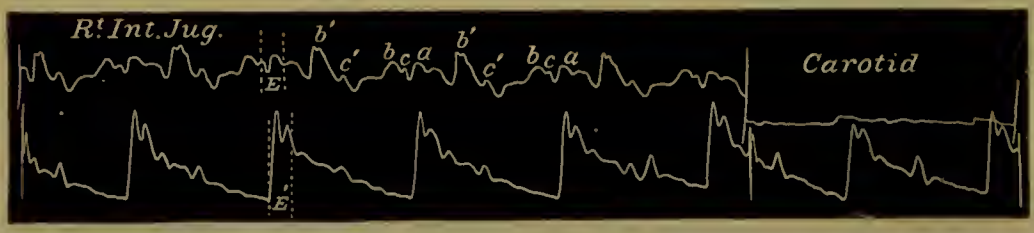

Fig. 110.—Simultaneous tracings of jugular and carotid pulses with the radial, during bigeminal action of the heart. The significance of the letters is the same as in Fig. 107 (Case 36).

only slight movements can be detected in them. There is marked pulsation in the deeper vessels of the neck, difficult to recognise by sight alone. The pulse is slow, about 50 per minute, but when compared with the pulsation of the neck there is noted a large pulse of the neck between the beats of the radial pulse. When the radial pulse is carefully palpated a very faint beat can be detected between the ordinary beats, and sphygmographic tracings show a pulse of the bigeminal type (Fig. 110).

The venous pulse taken at the same time shows a large elevation *b'* immediately preceding the small radial pulse. The ventricular wave appears only after the larger radial pulse, and presents a well-marked notch (pulmonary) on its summit, which corresponds in time with the closure of the aortic valves. There is only a slight cardiac impulse, and no defined apex beat. The heart's dulness extends $1\frac{1}{2}$ in. to the right and 5 in. to the left of the sternum. There is a systolic murmur heard loudest at the base (over both second costal cartilages and at midsternum); less distinctly at the apex and faintly in the axilla. The second sound is clear, and is immediately followed by two short sharp sounds, after which a pause occurs till the next recurring systolic murmur. The lungs are clear. The abdomen is enlarged, and contains a considerable quantity of fluid. The liver is not enlarged, and shows no sign of pulsation. The urine (about 40 oz. per diem) is deep coloured, and contains a large quantity of albumen.[1]

[1] This patient, since the above was written, has been tapped several times, and the ascitic fluid drawn off. On the last two occasions before the operation, tracings of the radial pulse, which was quite regular, were taken. Immediately after the tapping, tracings showed that the pulse had assumed the bigeminal form of irregularity.

The two foregoing cases exhibit the participation of the venous pulse in the bigeminal form of irregularity, but it is to be noted that the auricular wave corresponding to the small arterial beat is, as already pointed out, very large. In the following case, where also the venous pulse evidently presented the same form of irregularity as in the arterial pulse, the auricular wave corresponding to the small radial pulse is also the larger. The tracings here were taken separately, as no opportunity was afforded for obtaining a combined tracing. I employ it merely to draw attention to the increased size of the auricular wave *b'*, when it follows more quickly upon the preceding wave taken by a different instrument, to show that the peculiarities are not to be ascribed to a faulty instrument, and that they also occur when the irregularity is not constant.

Case 37.—Male, æt. 33; examined September 3rd, 1892; complains of weakness and indigestion. The patient is very soon tired, but has had a healthy life so far, and no serious illness. The heart appears quite normal. In the pulse there is an occasional irregularity; on a sphygmogram being

Fig. 111.—Sphygmogram of radial pulse, showing occasional irregularities of the bigeminal type (Case 37).

taken, shows it to be of the bigeminal form (Fig. 111). A long sphygmogram reveals this irregularity at irregular intervals, sometimes 20 to 30 beats intervening between the irregularities, sometimes only 2 or 3. A tracing of the venous pulse by means of the phlebograph shows that here

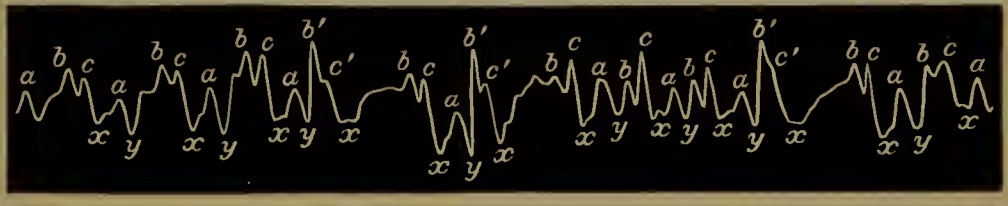

Fig. 112.—Tracings of pulsations in the right internal jugular vein, during occasional occurrences of the bigeminal form of irregularity in the heart's action. The irregularities are similar to those shown in Fig. 107, and the letters *b'* and *c'* indicate the auricular and arterial waves during the irregular periods (Case 37).

also the venous pulse evidently follows the radial. In Fig. 112 the first 3 beats were at the same rate as the preceding 20, taken on the long paper, the fourth beat follows rapidly on the third, then there is a high auricular wave *b'* followed by an interruption on the descent *c'*, which I take to be due to the arterial pulse; this is followed by a fall and gradual rise, which I presume to be due to the stasis preceding the next auricular wave *b*. In the tracing there are 3 beats presenting these peculiarities.

In the following case the venous pulse also accurately corresponds in rhythm with the arterial pulse. In this case it will be noted that the

radial pulse developed other forms of irregularity, and in Fig. 114 there is shown the occasional appearance of the lesser beat; probably when it is absent the incomplete ventricular systole has not sufficient strength to raise the aortic valves. I regret that I am not able to present a venous pulse synchronous with the radial pulse at this period. The patient had removed several miles into the country, and I called simply to observe the pulse; and as at that time the clinical polygraph was not constructed, I had to wait for an opportunity to employ the large recording apparatus, by which time a change had taken place, and the pulse had again become typically bigeminal (Fig. 115).

Case 38.—Female, æt, 34; examined first on May 5th, 1891; complaining of weakness and headache. The patient was confined of her fourth child 3 weeks ago, and appears to have had an attack of influenza a week ago. The pulse beats regularly, but each second beat is relatively smaller than the first.

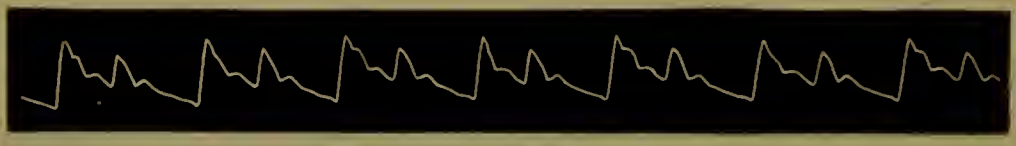

Fig. 113.—Sphygmogram of bigeminal pulse (Case 38).

A sphygmogram of the radial pulse shows it to be of the typical bigeminal form (Fig. 113).

The heart's position and size presented no abnormality. The sounds were free from murmur, but presented a peculiarity corresponding to the pulse, namely, after the first and second sounds of an ordinary beat there followed rapidly on the second sound two sharp sounds. (I question if I could have differentiated the sounds so exactly if I had not had the above sphygmogram of the radial pulse to guide me.) The following day the pulse presented the same peculiarity, no variation being detected. On the seventh and ninth the pulse presented a series of slow beats, with a short beat occasionally interpolated, and sometimes a few short beats occurring together. The patient was kept under observation for several months, and the pulse invariably presented the features of the pulsus bigeminus, as in Fig. 113. The patient removed to the country, and I lost sight of her for several months. In May 1892 she was in the eighth month of pregnancy, and the pulse was quite irregular, there being runs of small beats intermixed with the characteristic bigeminal form. I took a series

Fig. 114.—Sphygmogram showing mixed irregularities (Case 38).

of traeings on July 22nd, 1892. Some of these traeings showed a slow (44 beats to the minute) regular pulse; others showed a bigeminal pulsation intermixed, as in Fig. 114.

On September 21st, 1892, the sphygmogram showed a series of quick beats gradually passing into the bigeminal form, and also periods composed entirely of the bigeminal form. Traeings of the venous pulse taken at the same time, with the phlebograph, showed variations corresponding to the

pulse. These arterial and venous traeings were taken separately, but they bear out the interpretation of Fig. 115. Subsequent observations on the 3rd and 6th of November 1892 showed that the pulse maintained the bigeminal form. Fig. 115 was taken on the 6th of November from the left carotid and the right

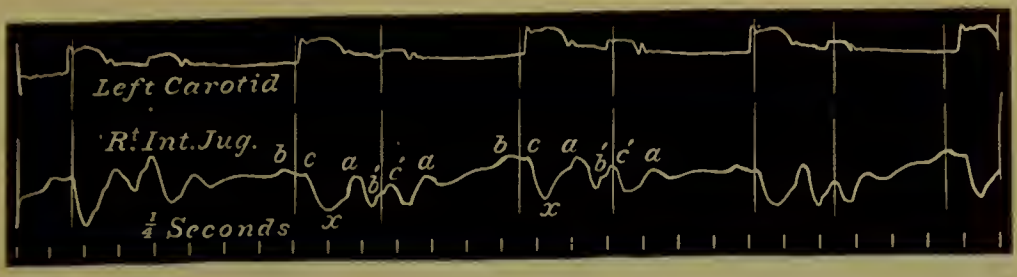

FIG. 115.—Simultaneous tracings of carotid and internal jugular pulses during bigeminal action of the heart. Corresponding with the lesser carotid beat there is a small auricular wave *b* in marked contrast with the large waves *b'* in Figs. 107, 111, and 112. Here there is also a ventricular wave *a* after the smaller arterial waves *c'*. Time in ¼ seconds (Case 38).

internal jugular vein at the same time. If the relative time of the events be carefully studied it will be found that the venous pulse follows accurately that of the artery—*b'* representing the auricular wave in the smaller beats, occurring as a low wave preceding that due to the carotid *c'*. During all the time that this patient has been under observation she has been able to go about and perform her household duties satisfactorily, nor did she appear to suffer from the effects of her confinement. (I did not attend her in her confinement.)

So far the irregularities of the venous pulse have corresponded with those of the arterial pulse. In Cases 35, 36, and 37 the auricular wave was unusually large at the period corresponding to the unusually small arterial pulse, and in Cases 35 and 36 there is no evidence of a succeeding ventricular wave. What these facts mean I cannot yet understand, and I merely call attention to them. In the succeeding 5 cases we have evidence of a discordance in the rhythm of the right auricular, and left ventricular contractions. (In all probability the auricles contract together and the ventricles together.)

CASE 39.—Female, æt. 53; examined May 1st, 1892; complains of great weakness, shortness of breath, and depression. The patient had rheumatic fever when she was 20 years of age, but has suffered from no serious illness since. She is a spare thin woman, with an anxious expression of countenance.

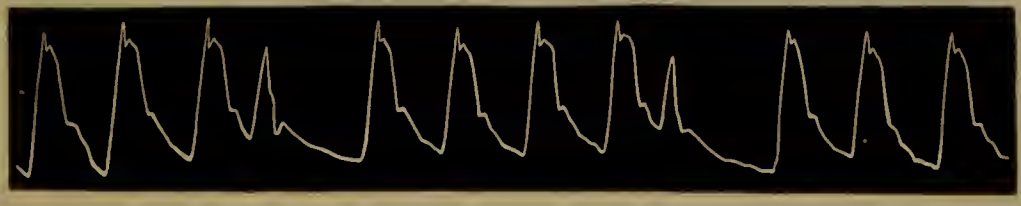

FIG. 116.—Sphygmogram of radial pulse, showing occasional occurrence of the bigeminal pulse (Case 39).

The radial pulse is large and full, and presents frequent irregularities. A sphygmogram shows these to be of the character of the pulsus bigeminus (Fig. 116). For a considerable period the irregularity occurs after every third beat, and then a long period may ensue with no irregularity. There is marked

capillary pulsation observed on the forehead after producing a faint blush by rubbing. There is also present a pulsation in the internal and external jugular veins. When the heart beats regularly this pulsation is very slight, but during the cardiac irregularity, and synchronous with the small arterial pulse wave, the jugular veins quickly pulsate to a great size. This large pulsation in the jugular vein is a very well-marked phenomenon, and could readily be timed

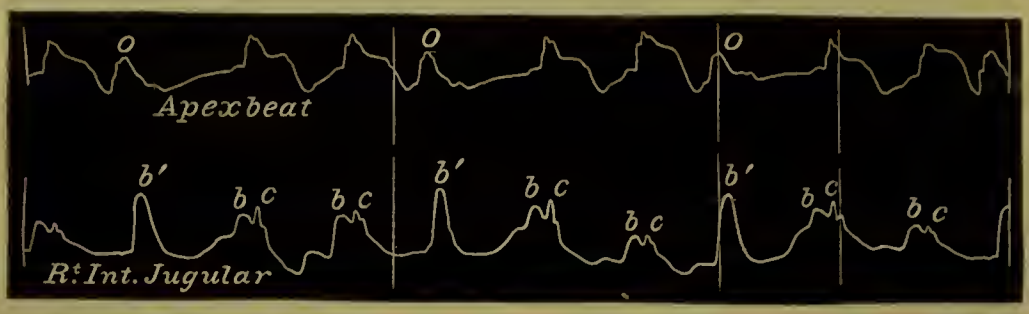

FIG. 117.—Tracing of apex beat (upper tracing) and of the right internal jugular pulse. In the apex beat every third beat (*o*) occurs earlier, is smaller, and is followed by a long pause. In striking contrast each auricular wave (*b* and *b'*) occurs at regular intervals. At the time of the small apex beat the auricular wave is large (*b'*), and the arterial wave is obscured by it (Case 39).

as occurring only during the irregularity. A large number of tracings were taken (now and also at subsequent periods), and they always presented the features present in Fig. 117, where during the small apex beat the venous pulse is very large. The heart is very slightly enlarged, and there is a diastolic aortic murmur, heard also during the irregularity, but of much shorter duration.

The confusing element here is the fact that the auricular wave *b'* occurs nearly at the same time as the ventricular systole and carotid pulse. In Figs. 118 and 119, *o*, there is shown a discordance in the time of the appearance of the carotid pulse, but not of the time in the appearance of the venous pulse. That the large wave during the irregularity was really venous could also be demonstrated by inspection of the veins. Whenever the small radial pulse occurred the jugular veins (which were easily recognisable in the spare neck) were seen to distend enormously. The extreme height of the auricular wave during the irregular periods I attribute to the following cause. When the auricle is contracting the ventricle is also contracting (the carotid pulse and the venous pulse appearing simultaneously); in consequence of this there is only one outlet for the auricular contents, namely, back into the veins, and therefore the venous pulse appears much larger. Such appears to be the only feasible explanation, but it scarcely applies to the large auricular wave in Cases 35, 36, and 37, though even in those cases there may have been a diminution in the receptive capacity of the left ventricle. The next case (40) appears to be of great interest, for if my interpretations of the curves be correct there is an actual occurrence of the auricular contraction after the ventricular systole, supporting the interpretation of Figs. 117 and 119. The venous wave in Fig. 118 occurs exactly at the time of the carotid pulse, and might therefore be assumed to be really due to the carotid. But as a matter of fact the phenomenon is such a

striking one to the naked eye that I entertain no doubt as to the correctness of the identity of this wave with the jugular pulse—an impression confirmed by two of my colleagues. It was not easy to get from this patient a tracing of the venous pulse, synchronous with the carotid, inasmuch as the head had to be held in such a position to get a venous pulse as rendered it difficult to get the carotid at the same time. In the Figs. 118 and 119 the venous pulse is taken at the

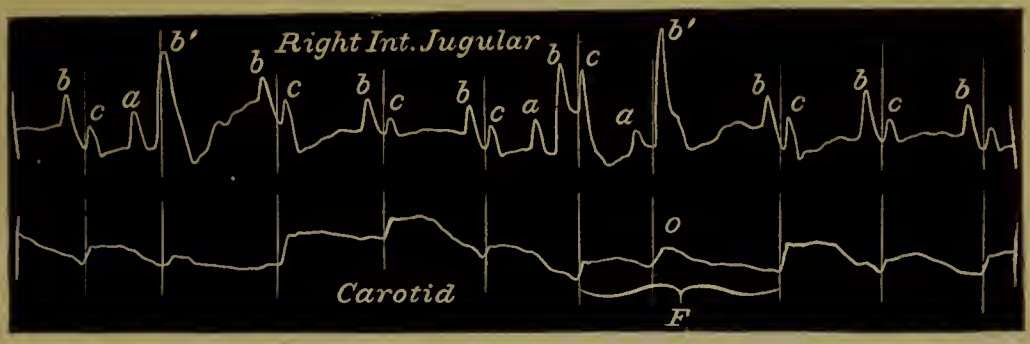

FIG. 118.—Tracings of the right internal jugular and carotid pulses taken together. The carotid pulse is occasionally irregular, but the auricular waves *b* and *b'* preserve their rhythm. The space *F* is unequally divided by the earlier occurrence of a small carotid pulse *o*, which is synchronous with the auricular wave *b'* in the venous pulse (Case 39).

same time as the carotid, and although they do not show the events very markedly, yet sufficient can be made out to recognise the independence of the auricular wave of the venous pulse and that of the small beat of the carotid during the irregularity. In many of the sphygmograms the interval between two regular beats exactly corresponds to that interval of irregularity during which the bigeminal pulse was shown. It frequently happened, however, that the small beat did not occur always at the same period of time after the large beat. Thus the

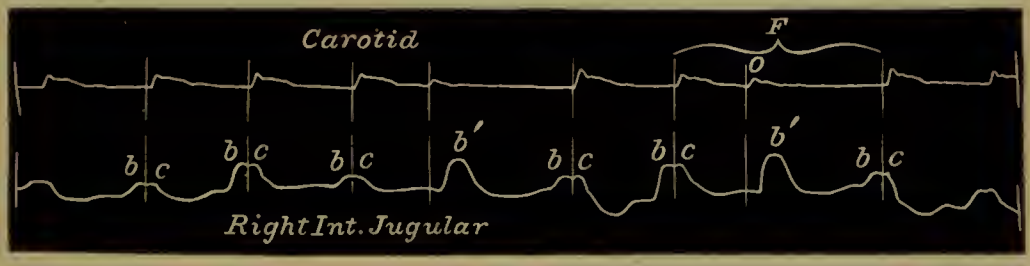

FIG. 119.—Tracings of the carotid and right internal jugular pulses. The waves *b* and *c* of the venous pulse really represent the auricular and arterial waves when the carotid is large. Where the carotid pulse occurs earlier it is smaller (Fig. 117) and does not show with the venous tracing. At such times the auricular wave *b'* occurs after the carotid, in contrast to what occurs in Fig. 118, when the small carotid pulse was synchronous with the auricular wave *b'*. The smaller carotid pulse *o* occurs relatively earlier than in Fig. 118, but the auricular wave preserves its rhythm (Case 39).

irregular interval *F*, in Fig. 118, exactly corresponds in duration with the irregular period *F* in Fig. 119. But the succeeding quick beat *o*

occurs a little later in Fig. 118 than in Fig. 119. On the other hand it will be found all through these tracings that the auricular wave *b* always occurs at regular intervals. This is very marked in Figs. 118 and 119, where in Fig. 118 during the irregular period *F'* the auricular waves occur synchronously with the arterial wave, while in Fig. 119 the auricular wave occurs a little later.

Trivial as this feature is, yet it forms an important link in the chain of evidence, showing the independence of the auricular and ventricular rhythms. There appears a great tendency on the part of the auricle to preserve a periodicity of action. If, for instance, the distance between the auricular waves *b* in Fig. 117 be measured, they will be found practically to correspond, while the irregularity in the apex beat tracing, and in the radial sphygmogram, demonstrates the extreme irregularity of the left ventricle. Here it would seem that there is evidence of the auricles contracting independently of the ventricle. It might be assumed that the wave is due to the right ventricle; but there can be little doubt that the wave is not due to the ventricle, inasmuch as there is distinct evidence of the auricle being active.

Case 40.—Female, æt. 27; has been under my care, at intervals, for the last 10 years. She has had, on the whole, good health, and was free from any disease. Eighteen months ago I attended her for an attack of influenza, and during convalescence she developed an occasional irregularity of the pulse—of the bigeminal form. This irregularity became less frequent, and disappeared after about a fortnight from its first appearance. She again consulted me at the end of April 1893, feeling weak and easily exhausted. The pulse again

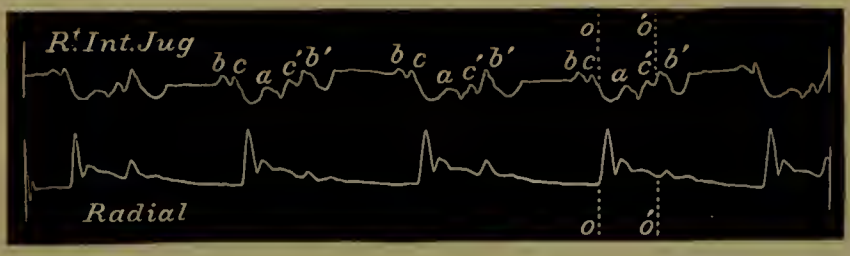

Fig. 120.—Tracings of jugular and radial pulses taken together. The waves *b* and *b'* are assumed to be auricular in origin, and occur at regular intervals, in contrast to the irregular rhythm of the arterial waves *c* and *c'*. The ordinates *o* and *o'* in the venous pulse indicate the same time as the ordinates *o* and *o'* in the radial pulse, showing that *c'* occurs at the same interval before the small radial pulse as *c* does before the large. (Case 40).

presented the same irregularity, the pulsus bigeminus continuing for some time. The patient says that she occasionally has uncomfortable sensations in the chest when these attacks come on, and she knows quite well when the heart is beating irregularly. There was only a faint pulsation in the veins, and I had some little difficulty in getting a good tracing. In Fig. 120 is given the radial pulse, taken at the same time as the pulsation in the right internal jugular vein.

It will be seen that the arterial wave *c* occurs a little before the radial pulse. This time corresponds with that taken from the carotid in other tracings. The small wave *c'* is thus recognised as the arterial

wave, preceding the imperfect beat in the radial pulse by the same interval as *c* precedes the larger radial beat. The wave *b'* I consider to be due to the auricular contraction, inasmuch as it preserves a uniform distance in all cases from the preceding undoubted auricular wave *b*. It is possible that the wave *b'* may, after all, be ventricular in origin, but, so far, we have seen no evidence of the ventricular wave occurring with the lesser beat of the bigeminal pulse, and there is no other wave that can be attributed to the auricle. I therefore incline to the view that we have here a preservation of the periodicity of action in the auricle while the ventricle was acting irregularly.

In only one case have I found this form of irregularity in a patient presenting a liver pulsation of the auricular type. I have been able to obtain a tracing from such a case, and the evidence here again supports the views already enunciated regarding the retention of its periodic activity by the auricle.

Case 41.—Female, æt. 41; examined on September 2nd, 1892; complains of weakness and shortness of breath. The patient has frequently been under my observation for the last 13 years. She suffered from rheumatic fever in her childhood, and when I first examined her 13 years ago the enlargement of the heart and other symptoms did not differ much from those here recorded. I have attended her for three attacks of rheumatic fever during the last 10 years. There has never been any irregularity until this examination. Now the pulse, every 20 or 30 beats, presents the form characteristic of the bigeminal pulse. There is marked heaving of the chest and a well-marked apex beat in the fifth interspace, with a distinct purring tremor preceeding the beat. The vertical dulness of the heart begins in the second interspace, and the transverse dulness extends from 1 in. to the right of the middle line outwards to the left for 5 in. There is a long murmur running up to the first sound heard only at the apex. There is a soft murmur following the first sound heard in the axilla. There is also a systolic murmur heard over the middle of the sternum and a faint diastolic murmur. The liver extends 2 in. below the costal margins, is tender on pressure, and pulsates distinctly (Fig. 122).

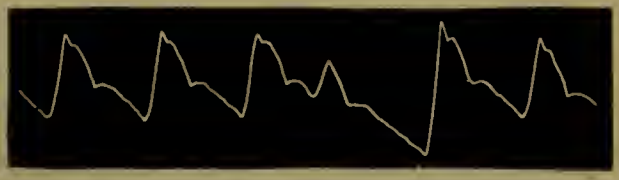

Fig. 121.—Sphygmogram of the radial pulse, showing the occasional appearance of an irregularity of the bigeminal form (Case 41).

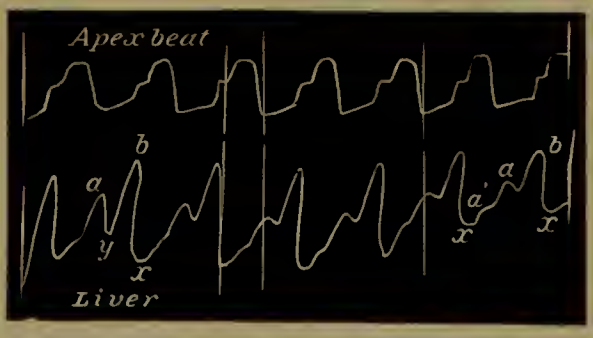

Fig. 122.—Apex beat and liver pulse (auricular type) (Case 41).

I found it necessary to take a large number of tracings before I succeeded in catching the pulsating liver during the irregularity. In Fig. 123 the occurrence of the irregularity is well shown, the regularity

of the auricular wave *b* being in marked contrast to the irregularity of the apex beat at *x*. The auricular wave during the ventricular irregularity is much larger than at other times, as in some of the foregoing cases. After this larger irregular wave also, there is complete absence of the ventricular wave *a*. There was only a faint venous pulse present, not of sufficient size to enable a tracing to be taken. When the patient was next examined, a week later, no irregularity was present, and the enlargement and pulsation of the liver had disappeared; when examined three months later, there was no return of the irregularity.

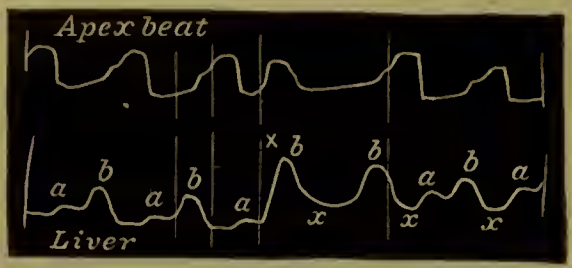

FIG. 123.—Simultaneous tracings of apex beat (upper tracing) and liver pulse (lower tracing), showing the bigeminal action of the heart *x*, with persistence of the regular rhythm of the auricular wave *b*, and absence of the ventricular wave *a* from the liver pulse after the lesser apex beat (Case 41).

The following case also affords some support to this view of the independent auricular contraction:—

CASE 42.—Male, æt. 49; examined December 27th, 1891; complains of weakness and shortness of breath. The patient has enjoyed good health until

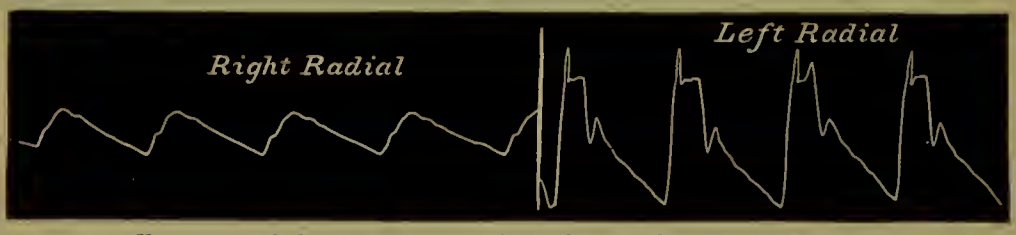

FIG. 124.—Sphygmogram of right and left radial pulse (Case 42).

the last few months. He is a powerfully-built man of medium stature. The pulse of the left radial is large and full and that of the right small (see Fig. 124).

There is marked pulsation at the sternal end of the sterno-mastoid muscle, and a tracing taken of this pulsation shows a large wave preceding the carotid pulse. There is no pulsation in the jugular veins. The chest is barrel-shaped and emphysematous, and the area of the heart's dulness cannot be defined with certainty. No pulsation is to be detected in the chest. In the aortic area there is heard a faint systolic murmur and an accentuated second sound. With rest the patient's condition improved, and he resumed his work. He was not seen again till the 21st of March 1893. He still follows his work, but is sometimes very short of breath. The relative size of the two radial pulses is still maintained, but now there is marked pulsation in both internal jugular veins, and in the right brachial vein to the bend of the elbow. The pulse is occasionally irregular (about every 20 beats), and the irregularity always assumes the form of the pulsus bigeminus, in the numerous tracings taken (Fig. 125).

The chest condition is much the same as at the first examination, save that the second sound at the base consists of two parts, the first short and soft, the second of a loud and booming character—the latter characteristic being heard over a great extent of the chest surface and into the carotids; at the apex there is a soft systolic murmur propagated into the axilla. There is no abnormal pulsation nor pressure symptom to be detected.

The features present in Fig. 125 of the venous pulse were those present in all the tracings. The chief point to be noted here is the

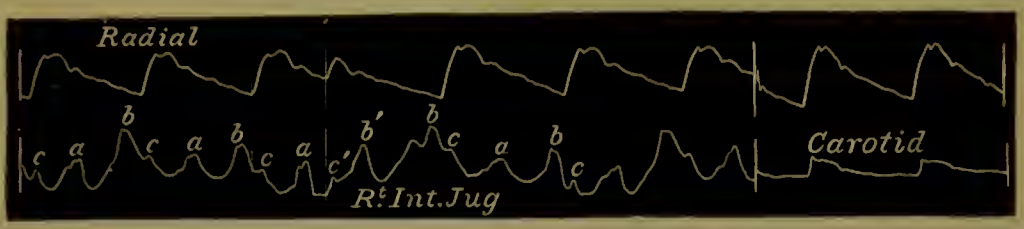

FIG. 125.—Simultaneous tracings of radial and jugular and of radial and carotid pulses. The small wave *c'* is assumed to be the arterial wave corresponding to the lesser of the two bigeminal pulsations, and bears the same relation to it as the carotid pulse and the arterial wave *c* does to the radial. The wave *b* is assumed to be an auricular wave (Case 42).

depression preceding the occurrence of the wave *c'*. On account of its time, I consider this wave to be the arterial wave. The wave marked provisionally *b'* is assumed to be caused by the auricular contraction, for the reason that the auricle has not contracted before the arterial wave *c'*, and because the distance which separates it from the preceding auricular wave is exactly the same as that which separates the waves when the pulse beats regularly. The wave succeeding it, however, occurs earlier, the distance during the irregular interval not occupying the time taken up by two pulse beats.

In the next case there was a frequent absence of an entire beat, the time occupied by the pause representing, usually, the time occupied by two pulse beats. Here, however, the auricular contraction maintained its normal periodicity. It may here be surmised, either that the ventricles ceased entirely to contract, or that their contraction was insufficient to raise either the aortic or pulmonary valves, as absolutely no sound whatever was to be heard over the heart during the pause. It is probable also that with increase of the strength of the heart the irregularity may assume the form of the pulsus bigeminus, by the interpolation of a rise in the diastolic descent of the tracing. (When the terms diastole and systole, unqualified, are used here, it is to be understood to refer to the periods of the ventricular action.) At times this patient's pulse only occurred at half its usual rate—every second beat being suppressed; but unfortunately, when this was observed, it was not convenient to take a tracing. I doubt not, however, that the auricle continued acting in the manner shown in the occasional irregularities. Another point to be observed here is the absence of the arterial wave *c* from the tracing during the pause—confirmatory to some extent of the view I have expressed relative to the causation of the arterial wave, inasmuch as its absence coincides with the absence of the carotid pulse.

CASE 43.—Male, æt. 54; complains of a disagreeable sense of oppression in his chest coinciding with a thumping action of the heart. The patient has

always enjoyed good health till the last few years, when he has been more breathless than usual on exertion. He is a medium-sized powerfully-built man. The pulse is large, tortuous, and full, the arteries somewhat atheromatous. At varying intervals there is a pause in the pulse beat, which usually beats

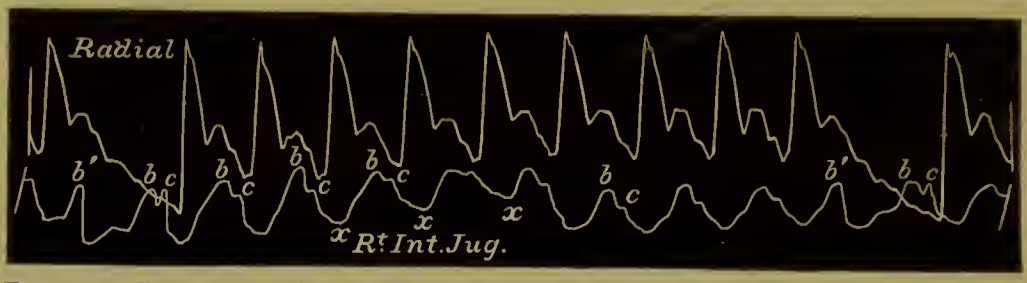

FIG. 126.—Traeings of radial and jugular pulses taken together, showing persistence of the auricular wave *b'* during a pause in the arterial pulse (Case 43).

about 88 per minute. Occasionally the pulse beats only between 40 and 50 times per minute, but only for 50 or 60 beats at a time. There is a slight pulsation in the veins of the neek, which continues during the cessation of the arterial pulse (Fig. 126).

The chest is large, capaeious, and barrel-shaped, respirations somewhat laboured—20 per minute. The lungs are markedly emphysematous, so that they entirely obseure the area of heart dulness. There is no heart impulse to be felt. The heart sounds are free from murmur, the first very faint and the second aeeentuated in the aortie area. During the pause in the radial pulse no sound whatever can be detected. There is no abnormality in any other organ.

Aside from the question of the independent contraction of the two sides of the heart, these last 4 cases demonstrate the independent contraction of the auricles. It is, of course, permissible to doubt the accuracy of the interpretation, and it may be possible to apply some other explanation. I may state that in each case a considerable number of tracings were taken, and they all showed exactly the same results. This dissociation of the auricular from the ventricular rhythm has been noticed before. Thus Chauveau[1] gives an account of a case of a slow pulse beating at the rate of 24 per minute; but there was manifested a pulsation in the jugular veins at the rate of 60 to 64 per minute, and in the tracings obtained, corresponding with these pulsations in the veins, there were slight elevations in the cardiogram, distinct from the well-marked apex beat. In this case he considered that the auricle was contracting more frequently than the ventricle. Experimentally, he demonstrated a somewhat similar condition of things by stimulation of the vagus. He assumed, and other elements in the case appeared to favour the assumption, that there was some irritation at the root of the vagus in his patient. Lépine[2] describes what he considers an anticipated

[1] Chauveau, A., "De la dissociation du rhythme auriculaire et du rhythme ventriculaire," *Revue de Méd.* 1885, p. 161.

[2] Lépine, R., "Sur l'eeartement des systoles auriculaire et ventriculaire dans eertains cas de bruits de galop," *Compt. rend. des séances et mémoires de la Société de biologie*, 1882, p. 97.

action of the right auricle, when there was a long pause between the auricular or ventricular systole.

So far the irregular rhythm presented by the bigeminal pulse has been studied in cases that presented the auricular form of venous pulse. When the same subject is considered in relation to the venous pulse of the ventricular form, less difficulty is encountered in the interpretation of the phenomenon, though there are still some interesting variations in the venous pulse. In Fig. 99 there has been noted an additional pulsation in the veins, when the left heart has assumed the bigeminal action. Usually, however, the rhythm of the right heart accurately follows that of the left. The pulsation of the vein generally alters its character. In place of the portion of the venous pulse that occurs after the closure of the pulmonary valves being the highest, as is generally the case, the earlier portion of the venous pulse becomes the highest, and the second portion may not be distinguishable. In Fig. 73, where the pulse presented a great variety of irregularities, some interesting features are to be noted. As a rule here, when a pulse beat is large, having been preceded by a longer pause than usual, the pulsation in the vein (or rather the jugular bulb) assumes some interesting peculiarities. The first portion of the venous pulse may be quite small or even entirely absent, while the second portion is enormously developed. On the other hand, when there are several small quick beats, the first portion becomes very large, while the second portion may be absent. But it will be observed that both pulses preserve exactly the same relative rhythm.

Case 44.—Female, æt. 32; examined May 7th, 1892; complains of swelling of the abdomen and shortness of breath. The patient had always good health until 3 years ago, when she began to get very short of breath on exertion, her condition gradually becoming worse. She is now unable to lie flat on her back,

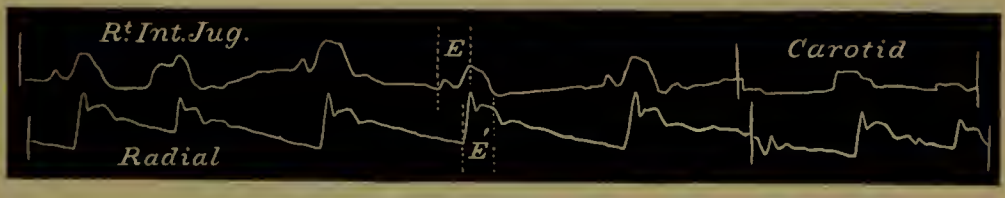

Fig. 127.—Simultaneous tracings of jugular and carotid pulses with the radial. The venous pulse is of the ventricular type, and its rhythm corresponds to the irregular rhythm of the arterial pulses (Case 44).

and her cough troubles her much. The patient's face is puffy and slightly cyanotic. When standing there is marked pulsation in the veins of the neck (Fig. 127), which pulsation almost disappears when she lies down, the veins then standing out full and tense.

The pulse is small and irregular, presenting long pauses, rapid series of small beats, and, occasionally, the typical form of bigeminal pulse. The heart communicates a sharp shock to the whole chest. The apex is felt in the fifth interspace; a fine purring tremor can frequently be felt preceding the apex beat. The vertical dulness of the heart begins at the second interspace in the left parasternal line, and extends transversely from 2 in. to the right of the middle line to 5 in. to the left. There is a murmur systolic in time

heard at the apex and in the axilla, and another of a different pitch heard over the lower part of the sternum. At the apex there is also heard a murmur during the diastole, but it does not run up to the first sound, and it is not heard at the base. The abdomen is distended; the liver dulness extends 2 in. below the ribs, and pulsates distinctly (Fig. 128).

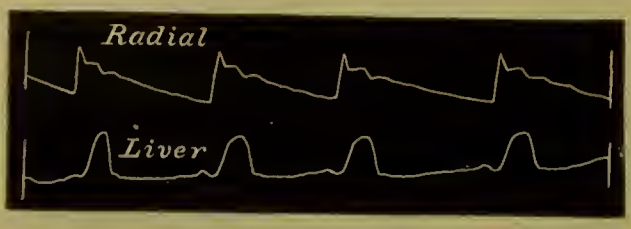

FIG. 128.—Simultaneous tracings of radial and liver pulses (ventricular type) (Case 44).

The percussion note elsewhere over the abdomen is resonant, but fluctuation can be detected. Since the first examination the patient's condition has undergone varying phases—sometimes improving, sometimes relapsing. Numerous observations have been made, and a great number of tracings taken of liver and venous pulses, with the apex beat, carotid, and radial pulses, and the same features have been present throughout as are manifested in Figs. 127, 128, and 129.

The pulse was always irregular, often markedly bigeminal, and the venous

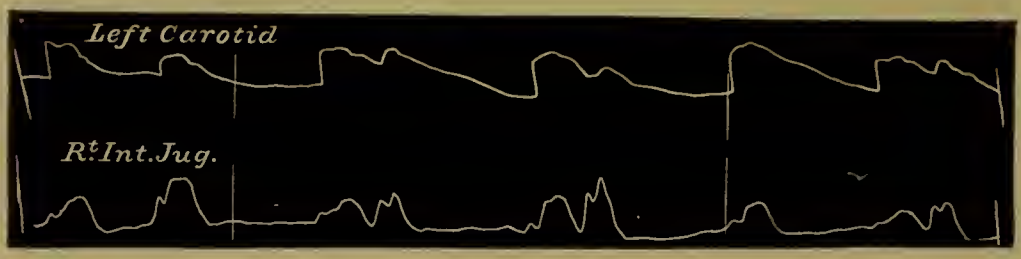

FIG. 129.—Simultaneous tracings of carotid and jugular pulses during bigeminal action of the heart, showing the agreement in rhythm of the arterial and venous pulsations (Case 44).

and liver pulsations always occurred rhythmically with the arterial pulse, as in Fig. 129.

The peculiarity in this case is a little wave always present in the venous pulse, sometimes not very distinct in the liver pulse, which precedes the main elevation. It has a quasi-resemblance to a small auricular wave, but if accurately timed it always agrees with the carotid

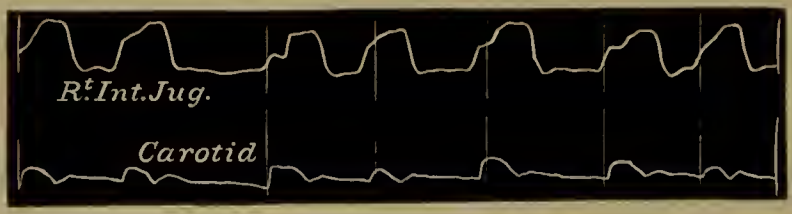

FIG. 130.—Simultaneous tracings of carotid and jugular pulses, showing correspondence in rhythm, but longer duration of the venous pulse wave (Case 44).

pulse in time (Fig. 130). The beginning of the larger wave succeeding this does not correspond to the closure of the pulmonary valves, nor is there ever any indication of the presence of this event. I presume that here, on account of the great distension of the venous system, as already

noted, the contracting ventricle throws such a large quantity of blood back into the veins that these are gradually distended. The continued outflow from the ventricle distends the veins to their utmost, so that when the pulmonary valves close the veins are maintained at the same height of distension. It will be recognisable at a glance that the duration of the venous pulse is much longer than the time occupied by the ventricular outflow through the arterial orifices. When one pulse succeeds another very rapidly, the small wave rises in height, sometimes very nearly to the level of the main wave.

Case 45.—Male, æt. 12. The report of this patient has already been published,[1] and I need only refer very shortly to the physical signs. There was a distinct history of chorea and rheumatism. There was evidence of great failure of the heart, the diagnosis pointing to stenosis and incompetence of the mitral

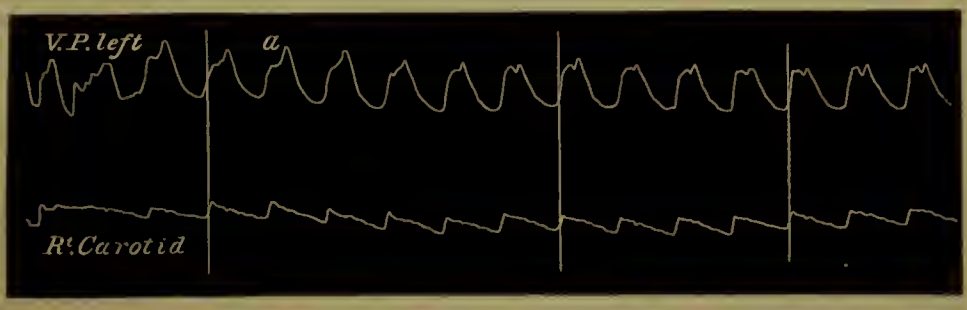

Fig. 131.—Simultaneous tracings of jugular and carotid pulses. The venous pulse is of the ventricular type (Case 45).

valves and incompetence of the tricuspid, with well-marked pulsation in the veins (ventricular type) and enlargement and pulsation of the liver (Figs. 34 and 37). The patient died on August 25th, 1892, and in the post-mortem examination the whole anterior aspect of the heart was made up of the right auricle and ventricle, both cavities being greatly distended; the right auricular appendix protruded under the second left intercostal space, just missing being pierced by a long needle inserted before the chest was opened in the second left space, half an inch from the sternum. The mitral orifice was narrowed, admitting one finger; the segments of the valves were slightly thickened and adherent at their free borders. The tricuspid orifice was greatly widened, easily admitting four fingers, and the right auricular appendix was greatly thinned. The pleural and peritoneal cavities contained a large amount of serous fluid, and the liver extended to the level of the umbilicus, and on section presenting characteristic nutmeg appearances of cyanotic atrophy.

On two separate occasions during the patient's life, while under the influence of digitalis, there was distinct irregularity of the pulse. The discordance between the venous pulse and the heart beat has already been commented upon in describing Figs. 99 and 100, which were obtained from this patient. Tracings of the liver and carotid pulses taken simultaneously during the occurrence of the bigeminal pulse do not show the peculiar rhythm so accurately as it appears in the venous pulse (Fig. 132); the third wave being absent from the carotid and

[1] *Journal of Pathology and Bacteriology*, vol. i. p. 76 (Case 8).

represented by a somewhat indefinite rise in the liver tracing, bearing, however, in some of the beats, a certain resemblance to the third wave

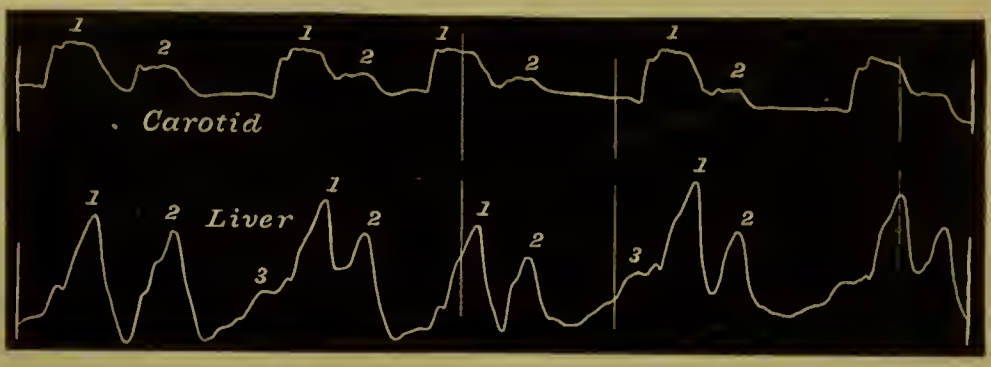

FIG. 132.—Simultaneous tracings of carotid and liver pulses (ventricular type). The carotid pulse presents the bigeminal rhythm, and waves 1 and 2 correspond to waves 1 and 2 in the liver pulse, while the imperfect liver wave 3 is absent from the carotid tracing (compare with Fig. 99) (Case 45).

in the jugular pulse (see Fig. 99). Occasionally the pulse was very irregular, and Fig. 133 shows the radial and liver pulses taken simul-

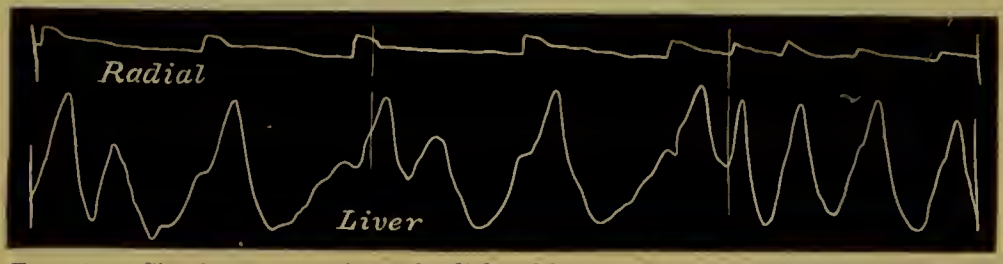

FIG. 133.—Simultaneous tracings of radial and liver pulses, showing greater frequency of the liver pulse (Case 45).

taneously, when several beats present in the liver pulse are absent in the radial.

CASE 46.—Female, æt. 30; examined July 2nd, 1892; complains of great weakness and shortness of breath. She is married and has 3 children, aged 11, 6, and 2 years respectively. She has had three attacks (never very severe) of rheumatic fever—12, 7, and 3 years ago; the joints were never very much swollen. Last November she had a good deal of pain in her feet, and she has been ailing ever since, with palpitation of increasing severity, breathlessness, and swelling of the feet. The pulse is quick, full, soft, and regular, 90 per minute. There is marked pulsation of the auricular type in the veins of the neck. On rubbing the forehead and producing a red blush, marked capillary pulsation is produced. The whole chest heaves with the force of the heart's pulsation. The apex beat is large and diffuse in the fifth and sixth interspaces near the anterior axillary line. The heart's dulness begins in the left parasternal line at the second interspace, and extends $1\frac{1}{2}$ in. to the right and 6 in. to the left of the middle line. At the apex there is a short murmur running up to the first sound, the latter being followed by a loud murmur heard round over the back on both sides. At the base there is a diastolic murmur, heard loudest over the second right costal cartilage, and propagated down the sternum. There is no increase in the size of the liver.

This patient has been kept under almost continual observation, and numer-

ous tracings have been taken of the venous and liver pulses. She has had several relapses, and during these the liver frequently descends as low as the umbilicus, and pulsates markedly. Usually the venous and liver pulses present the features characteristic of the auricular type, and have been used as illustrations in Figs. 4, 11, 28, 41, 45, and 52.

In these tracings of the liver pulse it will be observed that usually the auricular wave is larger than the ventricular, but I have occasionally found, when this patient is not so well and the symptoms of cardiac failure are more pronounced, that the ventricular wave *a* becomes the more pronounced feature, as in Fig. 134.

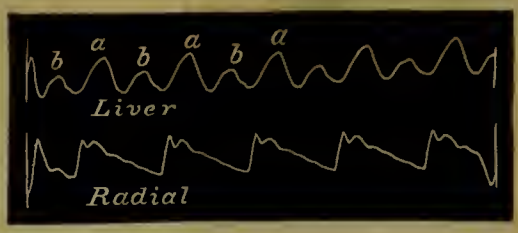

FIG. 134.—Simultaneous tracings of liver and radial pulses, showing the relatively larger size of the ventricular wave *a* (Case 46).

The tracing here represents the same features as the tracings in Figs. 56 and 78, from Cases 16 and 22, and, in this particular, forms a marked contrast to the usual liver pulse of the patient, as in Figs. 4 and 41. But the point most noteworthy in this case was a remarkable transient alteration in the respective rhythms of the venous, hepatic, and arterial pulsations. This occurred on two separate occasions, while the patient was recovering from severe attacks of cardiac failure. The first of these was noted on the 5th of September 1892. When examined on the 19th of August there was evidence of serous effusion in the left chest, and the liver was pulsating distinctly as low as the umbilicus. By the 5th of September she had markedly improved, the effusion in the chest was absorbed, and the liver still pulsating, projected but slightly from under the ribs. On this date I first took the tracing of the liver pulse, and the abdominal aorta at the same time (Fig. 45), which shows the usual rhythm. But while observing it, the venous pulse became very small and then increased in size. In order to appreciate the change that had taken place, I give here the usual rhythm of the venous pulse taken with the carotid (Fig. 135), which agrees in character and time with the tracing of the venous pulse in Figs. 4 and 11. The arterial wave *c* is here found to be exactly synchronous with the carotid pulse.

FIG. 135.—Simultaneous tracings of pulsations in the right internal jugular vein and carotid artery, showing the usual features of the auricular type of venous pulse with the auricular depression *x* during the beat of the carotid pulse *E* and *E'* (Case 46).

During the carotid pulsation due to the ventricular outflow, there is the usual well-marked fall in the jugular vein x, due to the auricular diastole

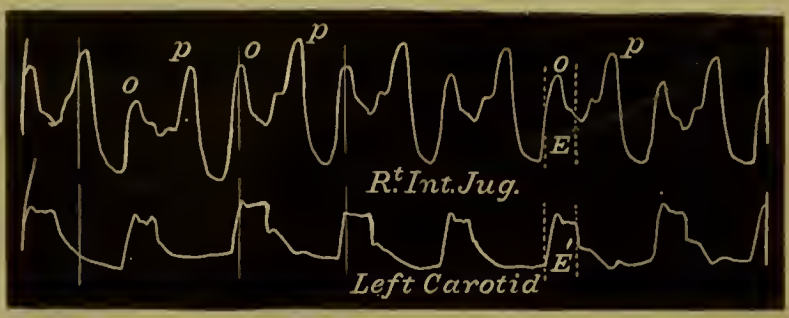

FIG. 136.—Simultaneous tracings of pulsations in the right internal jugular vein and in the carotid artery. The time of the events differs from those in Fig. 135, inasmuch as here there is now a high wave o during the period of ventricular systole E (Case 46).

E, lasting during the ventricular outflow E'. The other features are recognisable as those belonging to the auricular form of the venous pulse. But during this period of alteration of rhythm a marked change took place. Fig. 136 represents the carotid pulsation taken at the same time as the pulsation in the right internal jugular vein. It will be seen in this tracing that the period E' of the carotid pulse corresponds now to a period of elevation E in the venous pulse. The great depression in the venous pulse now occurs before, in place of during the carotid pulse and ventricular outflow. This difference is still more apparent in some other tracings. Thus in tracings taken on August 19th, when the patient's condition was very serious and the venous pulse well marked, the venous pulse presented a shape somewhat akin to that during the period of arhythmia. In Fig. 137, where the jugular pulse was taken at the same time as the apex beat, the period of ventricular outflow through the arterial orifices shows a depression E in the venous pulse during the same period of time in the apex beat E'.

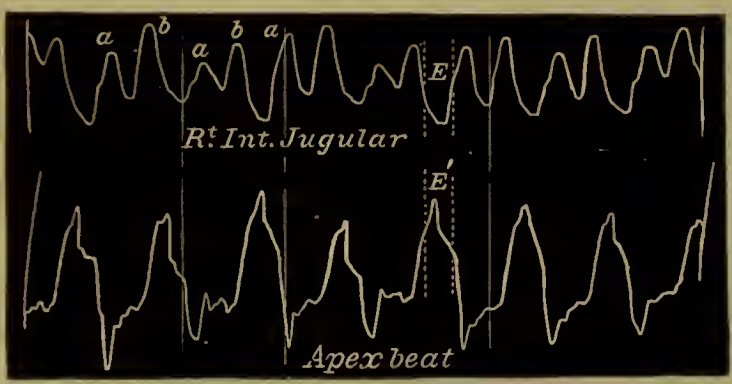

FIG. 137.—Simultaneous tracings of jugular pulse and apex beat, showing a depression in the venous pulse during the time of the ventricular systole E and E' (Case 46).

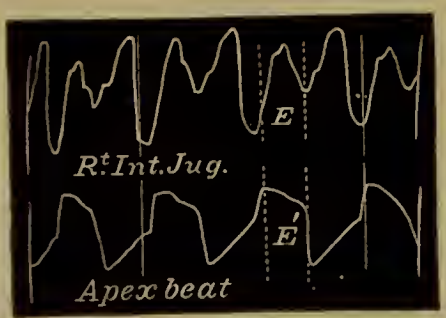

FIG. 138.—Simultaneous tracings of jugular pulse and apex beat, showing a wave in the venous pulse during the time of the ventricular systole E and E' (Case 46).

On the other hand, in Fig. 138, taken during the arhythmic period, the venous pulse shows a distinct wave *E* during the period of ventricular outflow in the apex tracing *E'*.

During this period of arhythmia the liver pulsation had also under-

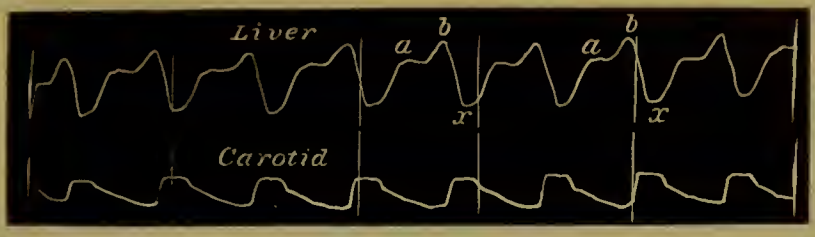

Fig. 139.—Simultaneous tracings of carotid and liver pulses (auricular type) (Case 46).

gone some peculiar change. Fig. 139 shows the usual form of the liver pulse taken with the carotid.

This was also the condition of the liver pulse when the first tracing was taken just before the arhythmia began (see Fig. 45, which was taken at this time). But the form of the liver pulse had to a great extent altered, as is shown in Fig. 140, where the carotid and the liver pulses are taken at the same time; and in Fig. 141, where the liver pulse and that of the internal jugular vein were taken together.

Fig. 140.—Simultaneous tracings of carotid and liver pulses during the arhythmic period (Case 46).

In this instance the peculiar arhythmic condition lasted for 2 days. During this time I took a large number of tracings. As the meaning of the change was at the time inexplicable to me (for that matter it is so still), I examined my apparatus carefully to see that it was not at fault, and took every precaution to ensure accuracy in the observations. For that purpose I took numerous tracings of all forms of combination of apex beat, carotid, venous, and liver pulsations, but all with the same result. On the 7th of September (2 days after the first appearance of that

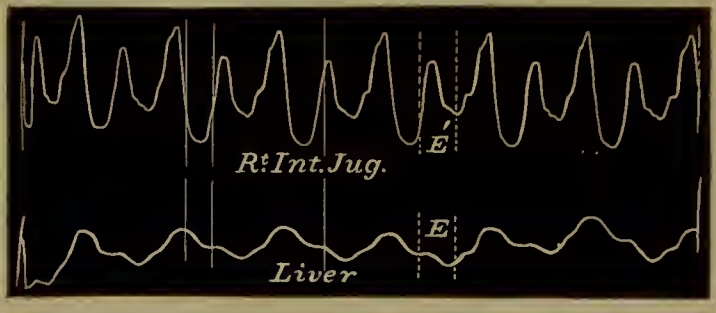

Fig. 141.—Simultaneous tracings of jugular and liver pulses during the arhythmic period (Case 46).

peculiarity) the usual rhythm was restored, and remained so, with one exception — when during a visit 2 months later the peculiarity was again present, but had disappeared the following day, and I have never observed it again.

The only suggestion I can offer for the explanation of this peculiar change is that either the relative rhythm of the two sides of the heart was altered or that there was a displacement of the time of the occurrence of the auricular systole. If we assume, for instance, that the wave *o*, synchronous with the carotid pulse in Fig. 136, is due to the auricle, then the beginning of the wave *p* would represent the time of closure of the pulmonary valves. That being so, the right auricle would contract synchronously with the left ventricle, and the right ventricle a little later than the left, and thus the wave *o* would represent the auricular wave *b*, and the wave *p* the ventricular wave *a* of the other tracings. If the other view be considered, that the wave *o* is really due to the ventricular systole, and therefore represents the ventricular wave *a* of the other tracings, it is possible that the ventricle, discharging itself into the auricle, stimulates it to contraction earlier than usual; hence it would contract immediately after the ventricular systole. That a certain amount of blood pressure in the auricle acts as a stimulant is accepted by Balfour,[1] Bramwell,[2] and others. In an experiment made by Sewall and Donaldson[3] it happened that the requisite pressure for stimulating contraction in the left auricle in the heart of a terrapin was acquired more rapidly than in the right, with the result that the left auricle contracted earlier than the right. On the other hand, it is expressly denied that the blood acts as a stimulus to contraction (Pettigrew,[4] M'William[5]).

After the foregoing was written this patient again came under my observation; the failure of the heart was so extreme that she died. During the last month of her illness the venous and liver pulsations presented further changes, which deserve some notice.

When examined on 30th May 1893, the position of the heart and the murmurs agreed with the description already given, except that there was now a marked systolic as well as a diastolic murmur in the aortic area. There was great œdema of the legs and trunk, but the lungs were clear. The pulse was quick and collapsing, 120 per minute. The veins of the neck were full, and gave rise to a tracing, mainly ventricular systolic in time (Fig. 142). The liver was enlarged and extended as low as the umbilicus, and some difficulty was experienced in taking

[1] Balfour, W. G., *op. cit.* p. 121.

[2] Bramwell, "Diseases of the Heart," Edinburgh, 1884, p. 11.

[3] Sewall, H., and Donaldson F., "On the Influence of Variation of Intra-Cardiac Pressure upon the Inhibitory Action of the Vagus Nerve," *Journal of Physiology*, 1880, vol. iii. p. 357.

[4] Pettigrew, J. B., "The Physiology of the Circulation, etc.," London, 1874, p. 328.

[5] M'William, J. A., "On the Rhythm of the Mammalian Heart," *Proc. Roy. Soc.* 1888, vol. xliv. p. 206.

a tracing, on account of the thickened abdominal walls. Numerous tracings were taken, and they always presented the features observed in

Fig. 142.—Tracings of jugular, carotid, and liver pulses, taken at the same time as the radial. *a* represents the ventricular wave, and *b* probably the auricular wave, and *x* the auricular depression (Case 46).

Fig. 142. Here it will be noticed that the main portion of the venous pulse *a* occurs at the same time as the radial pulse. There is a rounded elevation *b* after the descent has begun, followed by the great depression *x*. In the liver pulse the wave occurs practically at the same time as in the venous pulse; but the two waves are not clearly differentiated. It will be seen that in character and time the venous pulse here somewhat resembles the liver pulse in Figs. 140 and 141. The venous and liver pulses remained thus until 23rd July, when the pulse rate fell to 84 per minute, and the events in the jugular and liver pulses becoming more widely separated the individual features are more

Fig. 143.—Simultaneous tracings of jugular and radial pulses, showing small auricular waves and large ventricular waves (Case 46).

amenable to interpretation (Fig. 143). There is a small wave *b* immediately preceding the arterial pulse, and therefore due to the auricle. Following this is the ventricular wave, divided into two parts, one consisting of two wavelets near the same level as the auricular wave, and a high portion appearing after the auricle and great veins are fully distended. The wave due to the ventricular systole, after the closure of the pulmonary valves, is probably represented by the latter of the two small waves seen on the summit of some of the high waves. The radial pulse had become very small and weak, and there was some difficulty experienced in getting a tracing, and those obtained do not show the different parts with sufficient distinctness to enable me to speak with certainty.

The liver tracing shows practically the same features as the venous (Fig. 144), except that the auricular depression is more marked, and in consequence there is an absence of the first part of the ventricular wave.

Fig. 144.—Simultaneous tracings of liver and radial pulses (Case 46).

Next day the pulse had become very rapid, and both jugular and liver pulses presented characters similar to Fig. 142. On the 27th July the pulse was quieter, and the auricular and ventricular waves appear separate again, the highest wave being broader, and bearing two small waves separated by a slight depression, signifying, probably, the time of the closure of the pulmonary valves.

This patient's pulse hitherto had been always regular, but it so happened on the 23rd of June, when Fig. 145 was taken, while the patient was holding her breath, in order to facilitate the taking of a tracing of the liver pulse, there occurred a few beats presenting the

Fig. 145.—Simultaneous tracings of liver and radial pulses during bigeminal action of the left heart. The liver pulse maintains its uniform rhythm during the period of irregularity (Case 46).

bigeminal form of pulse (Fig. 145). The liver pulse is not very evident, but it can be observed that it maintained its wonted rhythm during the period of irregularity of the radial pulse.

On 30th June the patient's condition had become very serious. She suffered from great distress in breathing, with pain in the abdomen. The respirations were 52 per minute, there was incessant cough and bloody expectoration. There was dulness of the base of the left lung, accompanied by tubular breathing, and numerous moist râles. The pulse was 100, and the jugular pulse presented features similar to those seen in Fig. 144, except that the auricular wave disappeared during each respiration (Fig. 146).

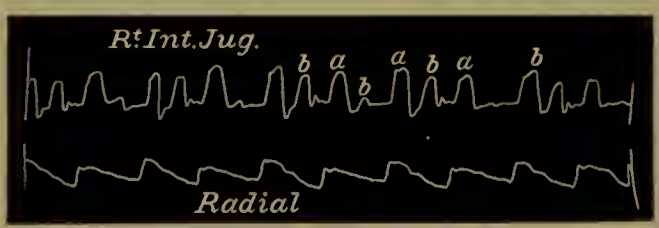

Fig. 146.—Simultaneous tracings of jugular and radial pulses, when the heart was rapidly failing. The auricular wave disappears in every second beat during inspiration. There are also changes in the radial pulse. Pulse 100; respiration 51 per minute (Case 46).

The patient died on the next day; at the post-mortem examination there was found great enlargement of the heart; the aortic valves were thick, shrunken, and incompetent. The mitral orifice was narrow, barely permitting the entrance of one finger, and the tricuspid valves were adherent, and the orifice narrowed, scarce admitting two fingers.

Section XX.—Adherent Pericardium.

That an adherent pericardium may exist and present no distinctive symptom is evident from the study of such cases as 18 and 19. In a case already reported by me,[1] a well-marked pulse of the auricular type was present during life, and at the post-mortem examination the pericardial sac was found to be obliterated. When, however, the external layer of the pericardium is adherent to the anterior thoracic wall (as in some cases of chronic mediastinitis), then it would appear that a venous pulse of a very peculiar and distinctive type occurs.

Friedreich[2] described the symptoms, and suggested a very ingenious and probable explanation. According to him the venous pulse is characterised by a sudden collapse during the ventricular diastole. The cause for this is explained as follows:—The adhesions between the heart and the anterior resilient chest wall drag in the chest during the ventricular systole. With the ventricular diastole the resilient chest wall suddenly rebounds outwards. This outward rebound straightens and depresses the diaphragm, and thus increases the size of the thoracic cavity. Consequently there is a sudden and considerable diminution of the intrathoracic pressure, and a lengthening of the superior vena cava, which result in the suction in of blood from the veins, producing in the jugulars a rapid diastolic depression. So far, however, I have not come across any tracings of the venous pulse demonstrating this particular phenomenon, and it must be a fact of rare occurrence, Friedreich's description appearing to be the only one; Sansom, indeed, is led to doubt the possibility of its occurrence.[3] The rarity of its occurrence must be my excuse for introducing the following somewhat imperfect tracing, but as it demonstrated, beyond the possibility of cavil, the fact noted by Friedreich it serves a useful purpose.

Case 47.—The patient from whom it was obtained had been under the care of Professor Leech of Manchester for nearly two years. The lad had suffered from undoubted symptoms of mediastinitis with adherent pericardium. The heart was enlarged, and there had been evidence of cardiac failure—œdema of the legs and enlarged liver. At one time there was present the typical *pulsus paradoxus*, with dilatation of the veins of the neck during inspiration and

[1] *Journal of Pathology and Bacteriology*, vol. i. p. 70 (Case 3).

[2] Friedreich, N., "Krankheiten des Herzens," 2nd edition, Erlangen, 1867, p. 129.

[3] Sansom, *loc. cit.* p. 97.

indrawing of the chest wall during ventricular systole. I saw the patient during a casual visit to the Manchester Infirmary, and with Dr Leech's permission I obtained the following tracing (Fig. 147) from the venous pulse at the same time as the radial pulse, by means of the clinical polygraph.

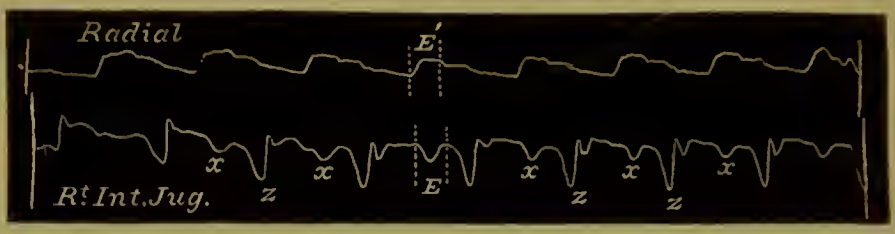

FIG. 147.—Simultaneous tracings of radial and jugular pulses, showing a great depression z during the ventricular disastole, from a case of adherent pericardium and mediastinitis (Case 47).

In the tracing it will be noticed that there are two main depressions, the smaller of the two (x) occurring during the period of the ventricular systole E and E'. There can be no doubt that this depression is due to the auricular diastole, and represents the usual auricular depression. The large depression z occurs immediately after the ventricular systole, and can only be attributed to the recoil of the chest wall after the systolic indrawing has ceased. The slight sharp wave immediately following the depression z is in all probability artificial, and due to the recoil of the membrane after the very sudden filling of the vein which takes place at the end of this depression. In some of the curves the auricular and arterial waves are faintly indicated.

SUMMARY.

The form the venous pulse assumes in the slightest degrees of cardiac derangement is that in which the movements of the auricle are added to the venous current; that is to say, there is a wave in the veins due to the auricular systole, and a depression due to the auricular diastole. There is usually present a smaller wave, the ventricular, in the mildest forms of regurgitation, occurring after the closure of the pulmonary valves.

With increase of regurgitation this ventricular wave appears before the time of closure of the pulmonary valves, but the latter portion of this wave is always the most pronounced, and the time of closure of these valves is indicated by a sudden increase in the size of the ventricular wave.

In some advanced cases of tricuspid regurgitation this earlier portion of the ventricular wave encroaches upon the time occupied by the auricular depression, and both auricular wave and depression become less marked as the ventricular wave increases, until finally the auricle ceases to impress its movements upon the venous current. The ventricular systole and diastole are then communicated directly to the venous

current, and produce the ventricular form of venous pulse. In the same manner the liver pulse may undergo variations, but the regurgitant effects are not evident in the milder cases, only occurring where there is some organic disease of the heart giving rise to forcible regurgitation. The liver pulse also assumes features similar to the auricular and ventricular forms of the venous pulse, with some slight modifications dependent upon the difference in structure.

There is also a movement communicated to the liver from the variations in size of the ventricular cavities. This movement, so far as I have been able to detect, is a diastolic depression and a systolic recession, and is, therefore, at variance with the time usually assumed for the occurrence of these movements.

When the heart's action is quickened there is a change in the form of the auricular venous pulse, consisting first in the disappearance of the period of stasis between the ventricular depression and auricular wave; and, second, in a disappearance of the ventricular depression, and a blending together of the ventricular and auricular waves.

There is some probability that the contraction of the superior vena cava may manifest itself in the venous pulse. There is evidence that the variations in the venous pulse may be useful to indicate variations in the blood pressure, certain influences being found to further the increase, and others to produce the disappearance of the venous pulse.

The observation of the venous pulse, with left heart action, serves as the best means of determining the lack of harmony between the contraction of the different heart chambers, and the cases of irregular heart action here recorded can be separated into seven groups.

1. Those in which there is complete agreement in relative rhythm during certain phases of irregularity of the right auricle and ventricle and left ventricle (Cases 31, 34, 38, and 44).

2. Those in which there is an exaggerated auricular impulse during this period of irregularity (Cases 35, 36, and 37).

3. Those in which there is a maintenance of the periodicity of action of the right auricle during irregular action of both ventricles (Cases 39, 40, and 41).

4. One in which there is a persistence of the auricular systole during a pause in the ventricular systole (Case 43).

5. One in which there is a contraction of the right ventricle during a pause of the left (Fig. 99; Case 45).

6. One in which there is complete discordance in the action of the two sides of the heart (Figs. 103 and 104; Case 18).

7. One in which there is a probable displacement of the relative time of the right and left heart movements (Case 46).

In conclusion, I have to acknowledge my indebtedness to my colleagues at the Victoria Hospital for the opportunities they have afforded me of studying cases under their care, and to Professors Roy and Sherrington for valuable suggestions.